# Modelling Graphemic Buffer Disorder:

# A connectionist approach

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I, Jonathan Machtynger confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

# Abstract

In studies of dysgraphia, graphemic buffer disorder is considered a distinct syndrome since traditional models of spelling place the graphemic output buffer at the junction of both the lexical and sub-lexical processing paths. Damage to the graphemic buffer often results in well recognised symptoms, but there is evidence to suggest that damage presumed to exist prior to the graphemic buffer such as with deep dysgraphia may also produce errors qualitatively similar to graphemic buffer disorder.

We build on an existing connectionist model of the graphemic output buffer and examine how damage inspired by physical impairment found in the nervous system may produce characteristics of both disorders. Since each has been observed to produce some common attributes, we investigate in more detail, and expand on, a claim made by others of a new putative functional syndrome.

As part of our investigations, we suggest a methodical and rigorous approach to lesioning connectionist systems. We also critique a number of core design principles associated with the original model and augment its functionality to allow a broader theoretical examination of a number of new areas. These include the production of geminate errors, whether orthography affects a word's propensity for error, and how the model may explain a minimum complexity principle associated with repair strategies in the presence of damage.

This thesis models disorders, which are known to produce subtly different, yet qualitatively similar behaviours in different patients, and we assume that a model must be able to produce comparable behaviour. In order to provide a rigorous and structured approach to analysing our results, we create a number of *quasi-patients* and examine the effect of damage across multiple lesion severities, lesion types, and lesion locations.

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# 1 Introduction

The fields of psychology and connectionism can be thought of as overlapping in many areas, and many researchers have used connectionist, or *neural*, approaches to model observable behaviour, which at a superficial level seem to reflect underlying cognitive processes. McCloskey (1991) makes an interesting suggestion regarding connectionism's place in cognitive science. Namely, that connectionist networks should not be viewed as theories of human cognitive functions, or as simulations of theories, or even as demonstrations of specific theoretical points. Rather, he argues, these networks hold considerable promise as tools for the development of cognitive theories. He may be asking "*How do networks of this sort contribute to our understanding of human cognition?*".

This thesis builds on existing connectionist networks modelling theories of serial order production, the simulation of spelling, and the analysis of patient behaviour related to the disorder of these tasks. Our model produce spelling errors analogous to those often found in Graphemic Buffer Disorder (GBD), Deep Dysgraphia (DD), and to a lesser extent unilateral neglect. In particular, we build on a model described in Glasspool, Shallice & Cipolotti (2006), which includes, refines, or modifies aspects of other functional models described in Glasspool, Houghton & Shallice (1994), Glasspool (1998), Ward & Romani (1998), Glasspool & Houghton (2005), and Burgess & Hitch (1992). As we based our work on previous efforts, we first reproduce many of the results described in the original publications, and then examine how the model improves upon that foundation. In order to do this, we developed an analysis workbench that allows us to evaluate both the model's performance as well as the performance of raw patient data, when it has been made available. There are a number of computational

methods for describing patient data in the literature, and the output of our model provides many of these. We are therefore able to compare various interpretations of relevant patient behaviour.

Chapter 2 discusses some of the theory underlying spelling production. The crux of the thesis revolves around understanding the behaviour resulting from intact and damaged graphemic buffers. Before we investigate this, it is necessary to articulate the theoretical foundation of a dual-route model of single-word production and situate how this dysfunction relates to damage associated with the graphemic buffer.

Chapter 3 investigates a proposal originally made by Cipolotti, Bird, Glasspool & Shallice (2004). They claim that deep dysgraphia may produce a qualitatively different form of graphemic buffer disorder occurring regularly enough to constitute a new putative function syndrome. Cipolotti et al differentiate between *classic* graphemic buffer disorder and their putative syndrome by terming them type A and type B graphemic buffer disorder respectively. We provide an extended literature review in the context of their claims, and examine each graphemic buffer disorder variant relative to patients with each disorder. We also highlight one possible extension to their claim by identifying an additional type A and type B symptom.

The physical nervous system is susceptible to all manner of damage resulting from disease and trauma. The effects of this damage may be seen over time in progressive disorders such as Alzheimer's and Parkinson's diseases, or almost immediately through the symptoms of strokes. In Chapter 4 we examine the effects of different types of damage on the nervous system insofar as they can be applied to artificial models of physical neuronal structures often called neural networks, or connectionist systems. Computationally damaging such models is not a new enterprise, and in this chapter we review various techniques for applying artificial damage, making

parallels where possible with biological damage. We also introduce a lesion type, which we believe is novel to connectionist networks.

Chapter 5 investigates and expands on an existing model of spelling production (Glasspool, Shallice & Cipolotti, 2006) used to produce various symptoms of GBD and DD discussed in Chapter 3. We expand on the model by exploring the effects of other serial production mechanisms (e.g. that of Burgess & Hitch, 1992), alternative semantic vector designs, adding a geminate facility, and refining aspects of the queuing mechanism. We also describe a possible mechanism for explaining how patients sometimes produce *unknown* letters, manifested by a blank space where a letter clearly belongs. Since Caramazza & Miceli's (1990) paper was originally published, computing power has improved considerably, and we have updated their scoring mechanism to cater for the automatic analysis of more complex errors. In addition, we outline our lesion methodology, which we believe provides a rigorous approach to damaging, and analysing the result of, our model. We also describe an alternative approach to balancing errors across five serial positions than the oft-cited Wing & Baddeley (1980) method.

In Chapter 6 we describe the effects of applying the lesion types outlined in Chapter 4 to the model, and the similarity of errors to those produced by patients. In particular, we examine how well the model behaves with respect to the symptoms described in Chapter 3 related to concreteness, frequency, the error distribution, and the serial position effect. There are different interpretations in the literature of the effect of orthography on error rates, and in Chapter 6 we investigate this in some detail. In order to quantify the effect of damage on error rates, we introduce a quotient measuring the consonant/vowel complexity of a word before and after damage. This enables us to

comment on patient behaviour, but also to make a comparative analysis against the model.

One notable addition we make to the Glasspool et al model is the ability to produce errors associated with double letters (geminates). Geminate errors are not simply random occurrences of extra or missing double letters as patients usually produce these in a predictable fashion. Geminate additions for example, are more typical in words already containing a double letter. In Chapter 7, we propose a number of mechanisms that progressively provide a framework for explaining geminate error behaviour in the context of the Glasspool et al's original model.

Finally, in Chapter 8, we provide an overview of the work, highlighting areas of success and detailing areas where the model could have provided a more robust explanation for patient data. We will also identifying possible topics of future research.

# 2 A General Model of Spelling

## 2.1. Introduction

Imagine a scenario where you answer the telephone and are asked to leave a message for a colleague saying that "Mrs Sczpoutchskey will call again at 6pm". Before you have time to ask for the spelling she hangs up. You quickly grab a pen remembering how the name was spoken (/tʃæˈpʊtski/) and write "Chapootsky". This is clearly a guess but quite close and you ask yourself why "Mrs Brown" didn't call instead. The name Sczpoutchskey is not a common name in England or you may have been able to come up with the exact spelling without as much thought as you did with the name "Brown". The fact that you provided a plausible spelling suggests that you have an ability to assemble what should pass for a phonological match to an unknown name. Conversely, if the name "Brown" had been given, you would have been likely to write the name perfectly despite "Braun", and "Browne" being phonologically identical. This also suggests a complementary ability to recall known spellings of words you have already been exposed to. Indeed, if the person at the other end of the phone had said /braʊn/ with a slight German accent, you may even have been tempted to ask whether it was spelled "Braun". These alternative choices form the crux of our discussions in this chapter, and have been described in the literature as *dual-route* approaches.

In evolutionary terms, spelling seems to be a recent *innovation* with major writing systems of the world originating roughly 5000 years ago. It is therefore most unlikely that writing is an *innate* skill, but something that has been achieved by the nervous system assembling complementary functions, socially moulded over the millennia to produce what we now call writing. If we assume the latter, then we may be able to ascertain what these complementary functions are by examining the behaviour of

normal and disordered spelling. At least as far as the English language is concerned, the sounds and spellings of words can be extremely inconsistent. Homophonic segments of words such as RAIN, REIN, and REIGN, as well as inconsistent words segments such as COVE, LOVE, and MOVE, demonstrate that pronunciation cannot rely on a simple phonological to orthographic conversion mechanism alone. Languages such as Italian and Spanish do not suffer from this difficulty since spelling is almost always predictable. This inconsistency in converting from sound to correct spelling has been taken as a justification for the necessity of a lexical spelling system (for English at least). How else could one explain being able to spell words such as YACHT and COLONEL correctly without resorting to a dictionary of sorts?

In this chapter, we examine the mechanisms involved in spelling, but identifying the specific processes involved in producing a series of letters can be difficult. One method of deriving these processes is to examine mistakes in spelling, rationalising that dysfunction through damage (or momentary lapses) may highlight the existence of a facility used to produce normal behaviour. One well-known study by Wing & Baddeley (1980) examined the spelling errors of undergraduate students. The errors made by these apparently good spellers might provide some insight into the underlying mechanism of writing, but far more can be learned from the behaviour of individuals with evident damage to the nervous system. In discussing reading, Ellis (1993:40) stated that "*When cognitive neuropsychologists investigate acquired dyslexia, their approach is not so much to ask which part of the brain is damaged in which form of reading disorder, but to ask which part or parts of the normal reading process have been damaged or lost. That is, they seek to explain different patterns of reading breakdown by reference to models of the normal, skilled reading process*". Although we are investigating spelling, the same principles apply, and evidence of damage to a working system may therefore

provide an indication of what may be contained within that system, and is an extremely powerful tool.

The 1980's saw many researchers creating models based on Morton's (1969) logogen model, which assumed that language production could be conceived of as having a functional architecture. Researchers evolved the logogen approach, and more evidence of patients with acquired disorders progressively provided support for the theory that missing abilities could be attributed to distinct processes in the model. One advantage of investigating acquired disorders over developmental disorders is that patients are assumed to have produced intact behaviour prior to the onset of the damage. With patients suffering from acquired dysgraphia for example, there may be evidence of premorbid skills in the form of correspondence (e.g. Folk et al, 2002), thus providing an opportunity to compare the effect of damage on writing.

In this chapter we will review a model of spelling that is generally considered to account for the spelling errors of a number of patients. The model is based on dual-route principles in the sense that spelling is assumed to be able to take more than a single pathway in producing a word's constituent letters.

## *2.2. Central and peripheral dysgraphias*

In describing reading disorders, Shallice & Warrington (1980) introduced a general terminology to distinguish between disorders affecting a patient's ability to obtain a satisfactory word-form, and those in which it is clear that damage to one or more of the routes has occurred. The first of these categories was termed peripheral dyslexias, and the second central dyslexias. This broad categorisation distinguishes between general loci of impairment (i.e. peripheral and central) and specific disorders (e.g. visual, surface and deep dyslexia). The same distinction can be applied to writing in which a peripheral dysgraphia suggests that a problem exists in articulating a word either orally or in

writing, and a central dysgraphia reflects damage affecting one or more of the routes leading to a problem with identifying the correct word to be spelled. We will clarify these with an example. Patient VB (Ellis et al, 1987) showed a clear difference in behaviour with oral and written spelling. While VB showed clear problems with written spelling, her oral spelling was excellent. Similarly, DS (Chialant et al, 2002) produced the same behaviour, orally spelling all words correctly, yet only spelling 7% of words correctly in writing. Another example is provided by OM (Miozzo & De Bastiani, 2002) who made substitutions involving pairs such as B-P, C-G, and M-N that were confined to handwriting and accurately formed. The fact that these letters were accurately formed suggested that the substitutions were not simply badly written (e.g. P as a badly written B), but deliberate. As we will discuss later, the visual similarity of the substitutions seems to rule out a deficit located at any level preceding and including the graphemic buffer. The fact that oral spelling was intact suggested a deficit at the level of letter forms in a given font or script. A complementary example is provided by the English speaking patient BRK (Cipolotti & Warrington, 1996) who showed better performance on written than oral spelling. If we assume that oral and written spelling use the same underlying mechanism to identify or assemble the word to be spelled then this suggests that for VB, DS, OM, and BRK, the central process by which the sequence of "abstract letter identities" is obtained seems to be intact whereas the mechanism for concretely realising that sequence of letters may be faulty. These patients showed evidence of a peripheral dysgraphia as the dysfunction is peripheral to the core spelling mechanism. If there was evidence to suggest that the underlying word identity or structure prior to production were damaged, then it might indicate that the damage could be located earlier in the spelling process and thus be called a central dysgraphia.

Barry (1994) distinguishes between central and peripheral disorders by referring to them as *spelling* and *writing* respectively. Spelling refers to the central ability to retrieve or to assemble an orthographic representation that is a coded sequence of letters. Writing on the other hand refers to the means whereby that orthographic code is translated into the production of ink marks on paper, or articulation of those constituent letters as words (e.g. /eɪ/, /biː/, /siː/ etc.). For patients VB, DS, OM, and BRK, the ability to spell and write clearly seems to rely on different functional processes. This seems intuitive, but is there a more formal approach for arriving at a similar conclusion with more evidence to substantiate that claim? In addition, how can one conclude which functional elements are responsible for which behavioural aspects of normal and impaired reading? In the next section, we look at a tool for analysing causality and interrelationships called dissociation.

## 2.3. Dissociations

In trying to isolate the locus of impairment in a functional model, it can be tempting to jump to conclusions as to the source of the disorder. As a general point Shallice (1988: p72) states, "*It is fallacious to assume that the existence of different predominant error types across patients necessarily implies that different underlying systems have been damaged, or that apparently qualitatively similar errors arise from the same cause*". This is a healthy approach as it is too easy to generalise about a facility that we know so little about (the human brain). It is true however, that lesions typically do not occur randomly. They often follow a pattern of location that is determined by certain neurophysiological constraints such as the positioning of cerebral arteries. This leads to both common occurrences of the same disorders, but also different disorders that may be the result of the same underlying (now dysfunctional) mechanism (e.g. Andrewes,

2001). If we therefore retain a healthy sense of caution, we might use principles of association, dissociation, and double dissociation (e.g. Shallice, 1988) to arrive at an informed conclusion. Let us describe each in turn.

With *associations* a patient X may be impaired for example, on cognitive task A, and task B. The two impairments are said to be associated since they are both present in the sample patient. This should be interpreted cautiously since the *co-variation* may be due to overlap in the anatomical distribution of cognitive subsystems, which are not necessarily functionally dependent (e.g. Saffran, 1982). *Dissociation* is more convincing. If the same patient X shows impairment on cognitive task A, but responds normally on task B, then the two impairments are said to be dissociated, because one is present and the other is absent in the same patient. Patients VB, and DS, for example show evidence of a dissociation between oral and written spelling. This again should be interpreted with caution since the demands made on the underlying subsystems may be different. Shallice (1998: 232) describes tasks such as reading individual words aloud and the Vocabulary subtest of the WAIS as being relatively insensitive to the effects of generalised neurological disease, whereas Block Design or Digit Symbol subtests of the WAIS are claimed as making more demands on the subsystems. Perhaps even more convincing are double dissociations where in addition to patient X mentioned previously, another patient Y is found with impairment in cognitive task B, but normal performance in task A. This suggests that tasks A and B rely on different underlying processes. Shallice (1988, 234, 235) suggests that the effects of resource artefacts can be discounted for the simple reason that if both tasks were critically dependent on one subsystem, then it would follow from a non-decreasing nature of performance/resource curves that irrespective of the specific form they take, that $R_X > R_Y$ for task A (where $R_X$ is the level of resource available to patient X), and $R_Y > R_X$ for task B. The two

statements are contradictory suggesting that the performance on the two tasks cannot be explained in terms of the operation of a single subsystem. Patients VB and DS for example, showed better oral than written spelling, whereas OM showed better written than oral spelling; a clear double dissociation of the articulation process and an indication that different mechanisms may be used in oral and written spelling. Another good example of double dissociation is provided by patients PR (Shallice, 1981) who was able to spell familiar words, but unable to spell unknown or *non-words*, and RG (Beauvois & Dérouesné, 1981) who could spell non-words but found difficulty with familiar words. With RG, the likelihood of being able to spell a word depended on how predictable the word's spelling from non-lexical phoneme-grapheme conversion processes was. This double dissociation may be seen as evidence that there are cognitive processes involved in spelling known words that are not necessary for assembling non-lexical spelling and vice-versa.

Such theoretical tools allow cognitive neuropsychologists to conceptualise the structure of healthy or normal cognitive systems. In chapter 5 and 6 of the thesis we describe a number of connectionist sub-networks each responsible for different aspects of spelling production, and join them in ways that may explain disorder in the presence of damage. A modeller might create a causal relationship based on the relative placement of functional subsystems. Figure 2.1 for example, shows how one might model mechanisms underpinning association, dissociation and double dissociation respectively.

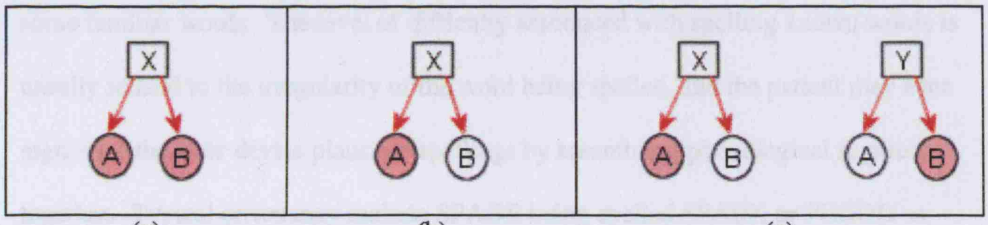



**Figure 2.1 : Possible mechanisms underpinning association, dissociation, and double dissociation viewed in terms of modular relationships between tasks A, B, C, and D.**

Let us briefly describe four disorders that will help provide the foundations for a further examination into what is a generally accepted model of spelling. We examine some of these in more detail in Chapter 3, but for the purposes of this chapter, they are convenient tools for helping us to situate the possible source of damage.

### 2.3.1 Phonological Dysgraphia

Phonological dysgraphia is a condition in which patients show a relatively well-preserved ability to spell familiar words but are very poor at reading non-words. This behaviour suggests that the patient has an ability to call on a so-called dictionary to produce the required spelling of familiar words. One of the first detailed examinations of phonological dysgraphia was provided for patient PR (Shallice, 1981) who was able to spell familiar words but had difficulty with non-words. PR seemed to use a compensation strategy by deriving phonemic segments for non-words from phonemic segments associated with real words. The non-word ITE for example was spelled HIGHT, and in correctly spelling UKE, he stated that he simply used the first three letters from UKELELE.

### 2.3.2 Lexical Dysgraphia

Unlike phonological dysgraphia, lexical dysgraphia (also known as surface dysgraphia) is a disorder where a patient is able to spell non-words yet shows difficulty in spelling

some familiar words. The level of difficulty associated with spelling known words is usually related to the irregularity of the word being spelled, and the patient may even regularise them, or devise plausible spellings by assembling phonological segments together. Typical errors may include SPADE being spelled SPAID, or FLOOD as FLUD as shown by patient PT (Hatfield & Patterson, 1983). Patient RG (Beauvois & Dérouesné, 1981) showed clear evidence of providing plausible spellings for non-words but difficulty in spelling known words with spelling accuracy being a function of orthographic ambiguity.

### 2.3.3 Deep Dysgraphia

Patients with deep dysgraphia (DD) show evidence of damage to both the semantic and non-lexical paths, and unlike phonological and lexical dysgraphia, DD patients exhibit difficulty spelling non-words as well as words. Typical symptoms include semantic errors such as writing CHAIR for TABLE, or BOAT for YACHT (Bub & Kertesz, 1982). There are also usually more errors associated with abstract than concrete words (e.g. Schiller et al, 2001, Nolan & Caramazza, 1983), and low frequency words than high frequency words (e.g. Orpwood & Warrington, 1995, Tainturier & Caramazza, 1996). In addition, words of different grammatical class (verbs, nouns, functors) may produce different error rates (e.g. Cipolotti et al, 2004, Ward & Romani, 1996). We investigate DD in much more detail in Chapter 3.

### 2.3.4 Graphemic Buffer Disorder

Graphemic Buffer Disorder (GBD) is generally associated with what are commonly considered 'spelling slips', as a consequence of combinations of letter substitutions, transpositions, additions, and omissions (e.g. Caramazza et al, 1987, Jonsdottir et al, 1996). Erroneous words usually exhibit more errors in medial serial positions than at

either end of the target word (e.g. Caramazza & Miceli, 1990, Hillis & Caramazza, 1989). In addition, longer words are usually more prone to errors (e.g. Miceli et al, 1985), and contrary to symptoms shown by VB and DS described earlier, the effect is present across all output modalities (e.g. Caramazza & Miceli, 1990). Unlike phonological, lexical, and deep dysgraphia, spelling accuracy is unaffected by lexical factors such as frequency, concreteness, grammatical class and spelling regularity (Caramazza et al, 1987). We also investigate GBD in much more detail in Chapter 3.

## 2.4 An introduction to the spelling model

At the beginning of the chapter, we described a fictitious scenario involving a message being left on behalf of someone whose name could be spelled in either a predictable or phonologically plausible way and we suggested that this may be attributable to alternative spelling strategies. This *dual route* approach to spelling has been discussed for a number of years (e.g. Ellis, 1982, Shallice, 1988, Ellis & Young, 1996, Tainturier & Rapp, 2001) and reflects the fact that a functional model of the spelling process may rely on alternative strategies depending on what needs to be spelled. We use the term dual-route loosely since this the model has also been described as having three routes. Figure 2.2 shows a generally accepted model of spelling providing multiple routes by which the spelling of a word can be achieved.

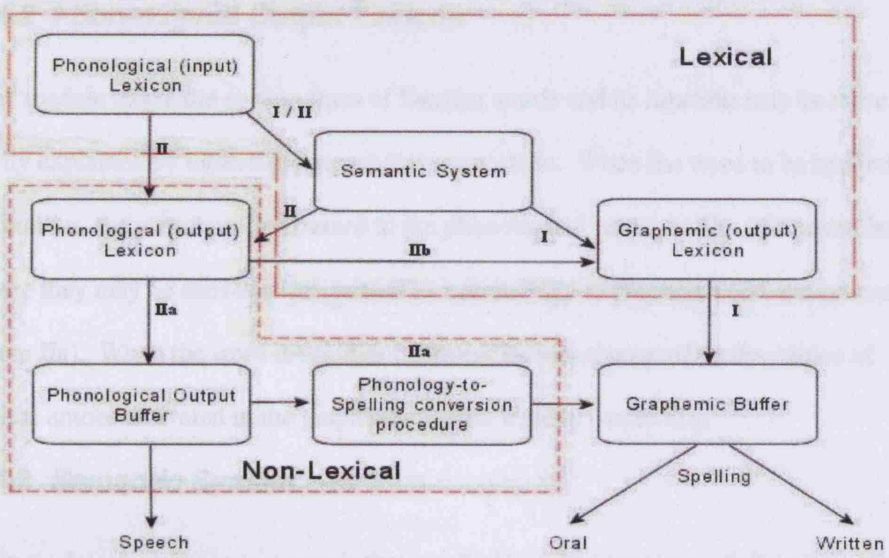



**Figure 2.2: Simple two-route model of the spelling process (Shallice, 1988). Route 'I' is the lexical/semantic route, and 'II' is the phonological route using sound-to-spelling transformations. Route IIa is the non-lexical phonological route, and route IIb is the lexical phonological route.**

The section labelled 'Lexical' can be used to spell known words. The non-lexical (or sub-lexical) route is taken where no record exists of the target word, and its spelling is assembled from its phonological form, which is more successful for words with a more regular than irregular phonology to orthography mapping. For the purposes of our discussion, we will use the term *module* to describe a self-contained functional process typically responsible for a single type of operation. We also assume however, that neuropsychological symptoms can arise as a consequence of damage to connections between modules as well as through damage to the modules themselves. Let us briefly examine the role of each module. Many do not directly take part in producing the four dysgraphias described earlier, but for completeness we provide a description.

### 2.4.1 *Phonological Input Lexicon*

This module acts as a spoken dictionary for the categorisation of a speech input as a familiar or unfamiliar word. Identified speech strings can use the lexical route whereas unidentified speech strings use the non-lexical route.

### 2.4.2 *Phonological Output Lexicon*

This module stores the spoken form of familiar words and its function may be more easily explained by understanding what it connects to. When the word to be spelled is unfamiliar, the phonemes are passed to the phonological output buffer (discussed later) where they may be subsequently passed to a phonology to grapheme conversion process (route IIa). When the word is familiar however, its *sound* may affect the choice of lexical entries activated in the graphemic output lexicon (route IIb).

### 2.4.3 *Semantic System*

This module contains known words that are distinguished by virtue of their semantic representation. Homophones such as SEEN and SCENE for example would both have distinct semantic identities. Semantic errors such as SKY → SUN (Bub & Kertesz, 1982) require more than damage to the semantic system alone; as we describe later in this chapter, damage to the non-lexical route (II and IIb) may be a requisite for such errors (e.g. Miceli & Capasso, 2006). Hillis et al (1999) also claim that damage to areas other than the semantic system may cause semantic errors. Patient RCM for example, produced semantic errors, in writing but not in reading. They conclude that this shows an intact semantic system and a joint deficit in 1) the non-lexical phonology-to-orthography mechanism (route IIa), and 2) access to representations in the graphemic output lexicon. They claim that an intact semantic representation may activate a number of semantically related lexical representations in the orthographic output lexicon, so when the target representation is unavailable due to impairment at the level of the graphemic output lexicon, the next most activated lexical representation is selected for further processing. It is unclear why semantically related items must necessarily collocate in the graphemic lexicon. We believe it more plausible that lexically similar entries would be more likely to collocate (e.g. Folk, Rapp & Goldrick, 2002). In

considering the relationship of the semantic system and type of semantic disorder associated with dyslexics (i.e. reading), Plaut & Shallice (1993) suggest that it is possible to classify patients as input, central, or output, based on whether their impairment is located prior to, within, or after semantics.

### 2.4.4 *Phonological Output Buffer*

This module maintains a buffer or temporary storage of phonemes for the desired word while it is either being spoken or assembled into written form. Damage to this system may result in substitution, deletions, additions, and transpositions of phonemes (e.g. Shallice, Rumiati, Zadini, 2000).

### 2.4.5 *Graphemic Output Lexicon*

Also known as the orthographic lexicon, this contains a dictionary of orthographic representations for known words. In languages with deep orthographies such as French or English, the proportion of words containing ambiguous or low-probability spellings is quite large. It therefore seems logical that in languages with shallow orthographies such as Italian and Spanish, the orthographic lexicon may be used less often than phonology to orthography translation. We will expand on the plausibility of this idea later in the chapter. For TP (Hatfield & Patterson, 1983) and RG (Beauvois & Dérouesné, 1981) the conventional spellings of some words were no longer available although they retained the ability to spell some irregular words, suggesting that the graphemic output lexicon though impaired was not entirely damaged. Note from Figure 2.2 that the graphemic output lexicon receives inputs from both the semantic system (I) and the phonological output lexicon (IIb). This reflects a belief (e.g. Ellis & Young, 1996) that the spelling of a familiar word is retrieved in response to a dual specification of its meaning and sound-form. Evidence for this *inner-voice* is apparently provided by *slips of the pen*, resulting in words such as THEIR being written for THERE, and SOUGHT

for SORT. A slip of the pen occurs when the writer knows how the word should be written and a momentary lapse results in an alternative spelling (of a real word). Spelling errors however, occur when a writer does not know the correct spelling of the word.

In a similar vein to Hillis et al (1999), Cipolotti & Warrington (1996) also suggest that the graphemic output lexicon may be responsible for semantic errors. Their patient BRK showed the classic signs of deep dysgraphia but did not make any semantic errors in naming, reading, and repetition tasks. They claim that this is consistent with a post lexical-semantic locus for the spelling deficit. Other authors (e.g. McCloskey et al, 1994, Folk et al, 2002) suggest that the graphemic output lexicon may be responsible for an effect of frequency. High frequency words are assumed to be represented more robustly in the graphemic output lexicon than low-frequency words, and thus be less susceptible to disruption.

### *2.4.6 Graphemic Output Buffer (GOB)*

Similar in principle to the phonological output buffer, this process maintains the activation levels of graphemic identities and sequences for the word being spelled deciding on the appropriate letter to output at a given time slice. Writing is a slow process, so Ellis (1993) claims a need for a short-term store capable of holding a word's spelling between retrieval and execution, and capable of retaining the latter portion of a word while the earlier portion is being written. Grapheme identities are held temporarily in this store as preparation for conversion into letter names (oral writing) or letter shapes (physical writing). Damage to the GOB is expected to affect familiar words and non-words since spelling knowledge will already have been formulated (from either or both the lexical and non-lexical sources). If both words and non-words are impaired to the same extent, then an obvious place to locate the damage within a traditional model is at

the level of the GOB itself. Similarly, an absence of semantic errors and symptoms involving imageability, frequency, and grammatical class would also suggest that the locus of impairment is likely to be in the GOB. Allographic details such as capitalisation, script, or case are held to be associated with subsequent processing (e.g. Ellis, 1982, Kay & Hanley 1994). Deficits should be also apparent regardless of how a word is spelled, for example spontaneous writing, writing to dictation, or delayed copy.

### 2.4.7 *Phonology to Spelling Conversion Process*

This process calls upon stored knowledge of the relationship between phonemes and corresponding orthography, and used for assembling an approximate form of an unknown word.

In summary, spelling can be achieved by retrieving stored spellings from the graphemic output lexicon (lexically), or by assembling and converting phonology into orthographic representations (non-lexically). It is assumed that until the graphemic output buffer is reached, that representations consist of abstract letters without a defined format. They can therefore be expressed in one of many modalities: oral, cursive writing, upper/lower case, typing etc. At a superficial level, damage to route I would lead to a reliance on the non-lexical route manifested as lexical dysgraphia by RG & TP, whereas damage to route II would lead to a reliance on the lexical route producing phonological dysgraphia such as shown by PR.

Tainturier & Rapp (2001) observe that many familiar words such as *cat*, *dog*, and *cup* are composed of common phoneme to grapheme correspondences and claim uncertainty whether the lexical route is strictly required, and Barry (1994) claims that regular words may be able to rely on simple phonemic to orthographic conversion. This seems implausible since homophones such as SEEN and SCENE are not interchangeable in normal prose, so verification of the semantic context of each word

must take place through the lexical route. An observation by Tainturier & Rapp of PR however, suggests that a single common path for unknown and regular-known words may not apply. Since PR spelled familiar words correctly, and there is evidence that the non-lexical route was damaged, then common regular words such as *cat* and *dog* cannot have been assembled phonologically.

## 2.5 Interdependence of Lexical and Non-Lexical paths

The existence of phonological and lexical dysgraphia may suggest that the lexical and non-lexical paths are completely autonomous, and may suggest, that at least in these instances, that there is no interaction between them. There is empirical evidence however that both paths do sometimes interact to some extent. Barry & Seymour (1988) for example, showed that the spelling of non-words appeared at times to be *primed* by the phonology of a word presented previously. If both pathways were completely independent, then the selection of constituent graphemes via the non-lexical phoneme-grapheme conversion process should be unaffected by prior activity involving the lexical pathway.

There is also evidence from phonologically plausible errors that partial lexical knowledge is sometimes available. Patient TP's errors of SWORD being spelled as SWARD and YACHT spelled as YHAGT suggests exactly this. A non-lexical phonological conversion is unlikely to have included the 'W'. Rather a more regular form such as SORD, or SAWED may have been produced. Something more elaborate than a simple lexical or non-lexical choice seems to be occurring here. As with PR's derivational strategy of assembling non-words through knowledge of real-words, TP may have assembled constituent phonemes together. The word WARD actually sounds more like *sword* than WORD does, so this may be plausible. However, the existence of the letter 'H' in YHAGT belies this, unless we assume that YHAGT involves a

transposition error with the word YAGHT. This seems to be a credible orthographic misrepresentation applied to a phonologically plausible error.

Miceli & Capasso (2006) discuss the relationship between phonological and orthographic lexical representations and between these and sublexical conversion mechanisms, converging towards the same conclusion - phonological and orthographic lexical representations are autonomous, but they may interact. They claim that damage to semantics or to output lexical representations is systematically associated with spared sublexical conversion procedures in subjects who do not produce such responses.

Miceli & Capasso (2006) describe how phonology may conceivably constrain the range of semantic paraphasias. The auditory stimulus /taigə/ for example, may activate incomplete semantic information (e.g. wild animal, feline, carnivorous, has fur, but not lives in Asia, fur is black and yellow striped), raising the activation level of orthographic forms sharing several of the conceptual features (e.g. lion, panther, tiger, leopard). If semantics were the only input to orthographic lexical representations, then any of these items could be selected for input to the graphemic output lexicon. In dictation tasks however, non-lexical conversion procedures work in parallel to lexical-semantic mechanisms probabilistically assembling a plausible orthographic string corresponding to the auditory stimulus. For /taigə/ therefore, *tiger*, *tigur*, and *tygur* are all plausible mappings, constraining the set of valid lexical values. Subjects producing semantic errors in spelling to dictation also demonstrate severe phoneme-grapheme conversion procedures. With no input from the phonological output lexicon (IIb), there is no *inner-voice* as Ellis (1982) describes it, to constrain the range of inputs to the graphemic output lexicon. In other words, without reinforcement of the sound /taigə/, then any of lion, panther, tiger, or leopard, all semantically close to tiger may be written in error. An interesting example concerns patient Jeannette (Hillis & Caramazza, 1995),

who showed improvement through explicit retaining of phonology to orthography. One indication that improvement was due to increased use of these procedures, rather than general improvement at the level of the orthographic output lexicon alone, was the continued presence of errors that were both phonologically and semantically related to the target, even though she stopped making purely semantic errors.

## 2.6 Conclusion

Our motivation in examining the general spelling model is to understand how patients with acquired language disorders can clarify the processes involved in intact spelling. Patients such as PR and RG seem to show evidence that the lexical and non-lexical routes are generally independent, yet there is also evidence to show that some form of interaction/integration between the routes exists beyond a mere competition for output. In this chapter we provided a very broad overview of a generally accepted model of spelling. It contains reference to a number of functionally specialised modules, each interacting to produce spelling, in either a written or spoken format. This chapter only provides an overview of these constituent modules, but for the purposes of the thesis, we believe it is critically important to situate the semantic, graphemic output lexicon, and graphemic output buffer functions in the context of the overall spelling model.

In the next chapter, we will examine symptoms of Graphemic Buffer Disorder and Deep Dysgraphia in substantially more detail investigating claims by Cipolotti et al (2004) that both disorders produce a subset of the same underlying symptoms. In doing so, we will also explore the nature of the GOB in much more detail.

# 3 Graphemic Buffer Syndromes

## 3.1 Introduction

As described in the previous chapter, spelling models can be quite complex, and damage in any one functional area may produce a wide variety of spelling errors. In this chapter we will concentrate on two specific disorders, namely Graphemic Buffer Disorder (GBD), and Deep Dysgraphia (DD). In both cases, patients produce spelling errors, each with different symptoms thought to be caused by suboptimal performance of the Graphemic Output Buffer (GOB) and Semantic systems respectively. Although we discuss how a functional breakdown of the semantic pathway can be recognised, our primary focus area is the GOB, assumed to maintain both the activation levels of letter sequences as well as a means of selecting the most appropriate order (e.g. Rapp & Kong, 2002).

## 3.2 Deep Dysgraphia

Deep dysgraphia can be considered an analogue to deep dyslexia. Traditionally, more effort has been invested in the analysis of reading than spelling, although spelling now seems to be receiving more attention. It should therefore not be surprising that symptoms of deep dyslexia (reading) were noticed first. Nolan & Caramazza (1983) presented data from patient VS showing that the defining symptoms of deep dyslexia could also be observed in any task requiring lexical mediation. Symptoms would therefore be very similar but applicable to spelling. Deep dysgraphia shows clear evidence of semantic effects with spelling errors such as TIME spelled as CLOCK (Bub & Kertesz, 1982), or FRIGHTEN as AFRAID (Nolan & Caramazza, 1983). In addition, evidence of difficulty in spelling non-words (as discussed in Chapter 2) implies damage to the non-

lexical route as well as the semantic system. Damage to the non-lexical route has been claimed as necessary for producing pure semantic errors (Miceli & Capasso, 2006). A word's imageability can also affect how prone it is to error. Patient VS (Nolan & Caramazza, 1983) and MRF (Orpwood & Warrington, 1995), for example, produced more errors on abstract than concrete words. Patient MRF, did not produce the effect with reading, suggesting dissociation in the type of damage between both patients as VS showed symptoms of both deep dyslexia and deep dysgraphia. Many patients show a clear difference in error rates between words of different grammatical classes, with nouns often being less error-prone than verbs, adjectives and functors, but this pattern is not always consistent across different patients. Patient PB (Schiller et al, 2001) for example, showed better performance on nouns and functors than verbs and adjectives, whereas DA (Cipolotti et al, 2004) showed better performance on nouns and adjectives than verbs and functors. Patients also show an effect of frequency with high frequency words showing fewer errors than low frequency words, and this effect may also apply to grammatical class. Patient DA for example showed fewer errors on concrete nouns than abstract nouns.

## *3.3 Graphemic Buffer Disorder*

Unlike deep dysgraphia, the effects of what we will call Graphemic Buffer Disorder (GBD) tend to manifest themselves as spelling mistakes with a predictable set of attributes: 1) Erroneous words show letter omissions, substitutions, transpositions, and inserts, or any combination of these, 2) A word's propensity for error is usually in proportion to its length insofar as longer words are generally more prone to error than shorter words, 3) Letters involved in a word's error are more likely to be situated in medial serial positions than at the beginning or end of that word, 4) Unlike deep dysgraphia, word frequency, grammatical class, and lexical status are not expected to

have any effect on error rate. Such symptoms were found to varying degrees in patients AS (Jonsdottir et al, 1996), BWN (Tainturier & Rapp, 2004), and ML (Hillis & Caramazza, 1989). Some patients did show aspects of frequency (e.g. DH – Hillis & Caramazza, 1989, HE – McCloskey et al, 1994), and lexicality (e.g. LB – Caramazza et al, 1987), but it is conceivable that the graphemic buffer was not the only part of the spelling system affected by damage. Each of these symptoms indicates that the graphemic buffer may be involved but as we will discuss further, a word length effect may also be produced for patients showing damage earlier in the spelling model.

The presence of spelling mistakes does not in itself suggest GBD. As discussed for example in Ellis (1983), chance mistakes, or *slips of the pen* provide evidence that apparently good spellers can often make inadvertent errors, such as writing THERE for THEIR.

## 3.4 The Graphemic Output Buffer

As described in Chapter 2, the spelling model (see Figure 3.1) assumes lexical and non-lexical pathways, with the lexical path using known spellings of words to help reproduce a desired written or oral spelling. The non-lexical path assumes no prior knowledge of a word's structure thus allowing a person to *spell* non-words, or unfamiliar words by converting phonology to orthography. In Figure 3.1 below, the non-lexical and lexical pathways are highlighted with red and blue pathways respectively.

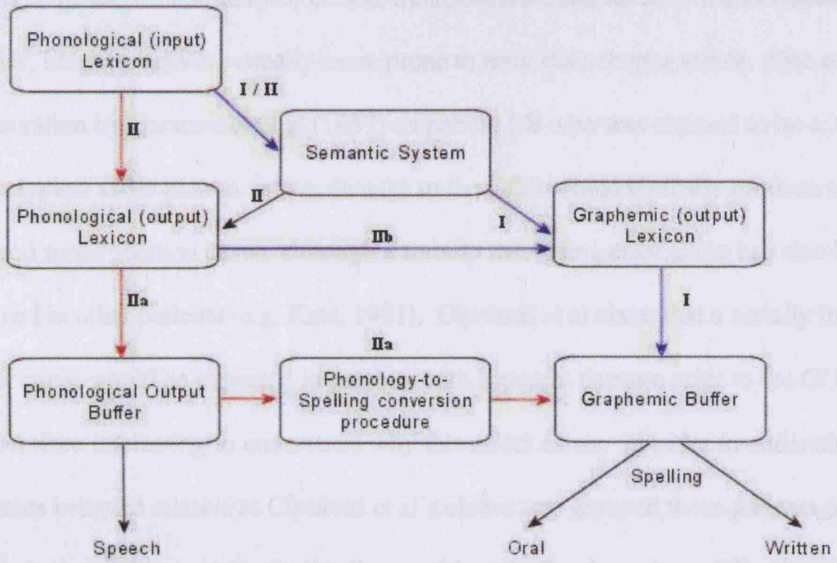



**Figure 3.1: A simple multiple-route model of spelling based on Shallice (1988). Route I is the semantic lexical route, IIa is the phonological non-lexical route, and IIb is the phonological lexical route providing sound-to-spelling transformations.**

The Graphemic Output Buffer is situated at the junction of the lexical and non-lexical pathways and can be thought of as a working memory system temporarily *holding* graphemic representations of a word during the execution of appropriate output processes. As described in Chapter 2, identifying the pathway of impairment may be possible by determining whether patients produce more errors on words or non-words. More errors on words than non-words suggests a problem in route I/IIb (e.g. Beauvois & Dérouesné, 1981), and in particular the effects of frequency, imageability, and grammatical class may highlight a problem at a more specific location within that path. More errors on non-words than words may suggest a problem with route I/IIa (e.g. Shallice, 1981). Key to understanding the nature of errors associated with these pathways is that the *absence* of errors affected by lexicality, frequency, imageability, and grammatical class suggests that the locus of impairment does not exist in either of these pathways, and is highly likely to exist at, or after the junction of both.

Since the GOB is considered responsible for retraining a word's constituent letters, their respective identities and serial order may be affected by damage, which is

likely to result in substitutions, inserts, transpositions, and deletes, and as discussed earlier, longer words are usually more prone to error than shorter words. One empirical observation by Caramazza et al (1987) on patient LB who was claimed to be a prototypical GBD patient, is that damage to the GOB would typically produce a bow-shaped serial position curve, although a serially increasing error curve has also been noticed in other patients (e.g. Katz, 1991). Cipolotti et al claim that a serially increasing error curve would be expected in patients with apparent damage prior to the GOB, and it is therefore interesting to understand why this effect exists. In order to understand how patients behaved relative to Cipolotti et al's claims, we grouped those patients according to their observable symptoms. As discussed later in the chapter, no difference in the error rates between abstract and concrete, or high and low frequency words is one indication that the GOB may be impaired. Core to Cipolotti et al's claim is that a functional relationship exists between patients who show damage *prior* to the GOB, such as those with deep dysgraphia, and certain symptoms typically associated with damage *to* the GOB, such as a consistent serial position effect. Cipolotti et al observed a pattern of behaviour between such patients, and their claims lay the foundations for a number of questions. For example, if the GOB is the likely source of symptoms linked to problems with letter order, then does evidence of damage presumed to exist prior to the GOB, and evidence of an intact GOB, indicate that a serial position effect is indirectly related to early damage?. A summary of symptoms produced by various patients is provided in Table 3.2, which we discuss in more detail later in the chapter.

## 3.5 Putative Functional Syndromes

The model in Figure 3.1 shows that the GOB sits at the junction of both the lexical and non-lexical pathways, and as previously examined, its theoretical role is to maintain the activation level of orthographic strings generated by activity from either or both routes

while sequential letter production takes place. As mentioned in section 3.3 above and expanded on further in the chapter, damage to the GOB is expected to result in a well defined set of symptoms such as for example, the word length effect and a predictable serial position error curve. This does not mean that such symptoms must therefore stem from a damaged graphemic buffer. Cipolotti et al (2004) have observed that deep dysgraphic patients sometimes produce GBD-like symptoms despite no apparent damage to the GOB and that behaviour shown by number of patients such as BA (Ward & Romani, 1996), DA, (Cipolotti et al, 2004), and TH and PB (Schiller et al, 2001), may imply a common *syndrome*. The term syndrome refers to a set of signs or symptoms that consistently appear together even though the source of that syndrome may be due to different loci of impairment. Cipolotti et al claim to have identified a new *putative functional syndrome*. Their term relates to a supposition that there is a common source for the syndrome's characteristics, that this is functional in nature, and that it relates to a putative (or assumed) area of the spelling system with its impairment being held accountable for the pattern of performance

This may be clearer with an example. A group of GBD patients such as LB (Caramazza et al, 1987), AS (Jonsdottir et al, 1996), ML and DH (Hillis & Caramazza, 1989) showed evidence of bow shaped serial position curves. A second group of patients showing evidence of semantic errors, and the effects of imageability, frequency, and lexicality (e.g. BA, DA, TH, and PB), did not show evidence of bow-shaped curves. The second group's symptoms suggest damage to routes I and IIb (from Figure 3.1) but do not necessarily exclude damage to the GOB itself. Of interest to Cipolotti et al, is that since serial ordering of a word's constituent letters is thought to depend critically on the GOB, and that the second group of patients produces a monotonically increasing serial position curve, then earlier damage may be indirectly responsible as a result of suboptimal input being passed to the GOB. One view on this is that degraded activation

reaching the GOB becomes evident through progressively more errors in later serial positions (e.g. Sage & Ellis, 2004). The lesion location, or *functional* source of the problem, may thus be held partly responsible for the symptoms produced. Cipolotti et al, claim firstly, that this causal relationship may be a candidate explanation for how errors are produced, and secondly, given the functional relationship of the semantic path to the GOB, that DD and GBD patients are likely to produce a number of common symptoms. As we will expand further in Chapters 5 and 6, the aim of our simulation model will be evaluate the plausibility of such a theory, and how such a functional relationship affects those symptoms considered to be core to graphemic buffer disorder. One way of thinking of these variants of GBD symptoms are "classic GBD" and "DD *related* GBD" syndromes, and Cipolotti et al. categorise them as GBD type A and GBD type B respectively. Table 3.1 shows a simple grouping of those behaviours typical of type A and type B patients. In short, type A behaviour reflects symptoms usually seen in GBD, and type B behaviour reflects symptoms that Cipolotti et al claim would be common in patients with DD. One of these error types, *fragments* (Ward & Romani, 1998) represents a word whose resultant length is at least two characters shorter than that of the target word.

| GOB Type A Behaviour | GOB Type B Behaviour |
|---|---|
| Show a bowed serial curve with lower error rates at the start and end of words | Error rates increase monotonically from start to end of word |
| Produce a majority of letter substitutions | Produce a majority of letter deletion errors |
| Fragment errors not observed | Produce large numbers of fragment errors |

**Table 3.1: A simple categorisation of type A and type B behaviours (Cipolotti et al, 2004).**

Cipolotti et al. claim that type A behaviour results from damage or disruption to the GOB in the presence of an intact semantic system whereas type B behaviour results from an intact GOB system operating under degraded input from the semantic system. Table 3.1 outlines a simple classification of each group of error types. Type A behaviour is assumed to stem from damage to the GOB itself whereas type B behaviour is assumed to

stem from an overall degradation of correct letter activation within the GOB. The observation that both types result in the mis-selection of desired letters suggest that the GOB is involved, but to varying degrees and it is this variance that defines the type A or B categorisation. One theory of how damage early in the spelling model may affect the GOB is given by Sage & Ellis (2004) as an observation of some of the errors made by their patient BH and Ward & Romani's (1998) patient BA - "*reduced activation of graphemic representations could mean that there may sometimes be only sufficient activation to drive the production of the first few letters, resulting in incomplete fragmentary responses*". Cipolotti et al seem to have generalised Sage & Ellis's theoretical claim in a framework encompassing a broader range of symptoms.

Cipolotti et al conclude that while DD and GBD seem to be distinct disorders, DD may affect the performance of the GOB, and thus produce variant GBD symptoms. This seems logical given that the GOB is situated *downstream* from the semantic system. We would claim that any damage located earlier than the GOB is highly likely to result in a number of GBD-like errors due to degraded input to the GOB. Indeed, we identify a further possible GBD-like symptom, which we believe may be indicative of damage in the lexical-semantic (I/IIb) path, namely a shallower word-length error curve, which we discuss in more detail in section 3.6.2.1. In Chapters 5 and 6 we will investigate the applicability of Cipolotti et al's claims, expanding on a model of the GOB put forward by Glasspool et al (2006).

## 3.6 Symptoms of Graphemic Buffer Disorder and Deep Dysgraphia

In order to provide an overall view of the typical behaviour of both disorders, we have summarised the symptoms of 22 patients (Table 3.2). The choice of patients originally stemmed from those in Sage & Ellis (2004) but some of the patients they considered were not included due to the unavailability of journals. We however, added a number

of other patients who showed symptoms associated with one or both of DD and GBD. It is important to note that we have not categorised our patient list into distinct DD and GBD groups. Primarily because many patients such as FM[1] (Tainturier & Caramazza, 1996) show symptoms common to both disorders and it would therefore be debatable to which group the patient rightly belonged. Rather, we categorised patients into two groups in which their dominant symptoms suggested damage to a presumed location within the theoretical model shown in Figure 3.1. This approach may seem too theoretical given that we are discussing symptoms associated with disorders. We would however argue that investigating a putative functional syndrome must consider what effect damage has on downstream processing as suggested by the theoretical model, and we must therefore consider reasons *why* these symptoms may be produced in order to categorise them appropriately. In Table 3.2 below, we subdivide the patients into two major groups, GOB and NON-GOB. These terms will be expanded on in more detail in the following pages, but as the groupings suggest, we believe that patient symptoms result from damage *to* the GOB, or to damage *earlier* than the GOB respectively. We would also expect that these groups will *broadly* map onto Cipolotti et al's type A and type B groups respectively.

[1] Considered to have a combined impairment of 1) the GOB, and 2) the lexical–semantic component and/or orthographic output lexicon, and 3) the phonology to orthography conversion mechanism

| | | Patient | Reference | Lang. | PPE | Gramm. Class | Sem. Error* | Imageab. | Freq. | Lexic. | Serial Curve | Word Length | *m* | Single Error Distribution |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| GOB | | SE | Posteraro et al (1988) | Ita. | (NR) | (NR) | NO | NO | NO | NO | Λ | YES | (NR) | DS |
| | | FV | Miceli et al (1985) | Ita. | (NR) | (NR) | NO | NO | NO | NO | -† | YES | (NR) | SIDT |
| | | LB | Caramazza et al (1987) | Ita. | (NR) | NO | NO | NO | NO | YES | Λ | YES | 0.171 | DSTI/SDTI‡ |
| | | DH | Hillis & Caramazza (1989) | Eng. | NO | (NR) | NO | NO | YES | NO | Λ | YES | 0.214 | DSTI |
| | | ML | Hillis & Caramazza (1989) | Eng. | NO | (NR) | NO | NO | NO | NO | Λ | YES | 0.157 | DTSI |
| | | AS | Jonsdottir et al (1996) | Eng. | YES | NO | NO | NO | NO | NO | Λ | YES | 0.174 | SITD |
| | | BWN | Tainturier & Rapp (2004) | Eng. | YES | NO | NO | NO | NO | NO | Λ§ | YES | 0.093 | DSTI |
| NON-GOB | NON-SEM | JH | Kay & Hanley (1994) | Eng. | YES | NO | NO | NO | YES | YES | (NR) | YES | 0.043 | SDIT |
| | | HE | McCloskey et al (1994) | Eng. | NO | NO | NO | NO | YES | NO | (NR) | YES | 0.114 | SIDT |
| | | JES | Aliminosa et al (1993) | Eng. | (NR) | NO | NO | YES | YES | YES | Λ | YES | 0.129 | (NR) |
| | | AM | De Partz (1995) | Fr. | (NR) | NO | NO | YES | YES | YES | Λ | YES | 0.139 | DSIT |
| | | HR | Katz (1991) | Eng. | (NR) | YES | NO | (NR) | (NR) | YES | / | YES | (NR) | Mostly Deletes |
| | | BH | Sage & Ellis (2004) | Eng. | YES | (NR) | NO | YES | YES | YES | Λ | YES | 0.199 | DSIT/DSTI** |
| | SEM | DA | Cipolotti et al (2004) | Eng. | YES | YES | YES | YES | YES | YES | / | YES | 0.042 | SDIT |
| | | FM | Tainturier & Caramazza (1996) | Eng. | YES | YES | YES | (NR)†† | YES | YES | Λ | YES | 0.117 | SDTI |
| | | VS | Nolan & Caramazza (1983) | Eng. | NO | YES | YES | YES | YES | YES | (NR) | (NR) | (NR) | SDIT |
| | | BA | Ward & Romani (1996) | Eng. | YES | YES | YES | YES | YES | YES | / | YES | 0.051 | DSIT |
| | | MRF | Orpwood & Warrington (1995) | Eng. | (NR) | YES | YES | YES | YES | (NR) | (NR) | (NR) | 0.048 | SDTI |
| | | JC | Bub & Kertesz (1982) | Eng. | YES | YES‡‡ | YES | YES | (NR) | (NR) | (NR) | (NR) | (NR) | (NR) |
| | | BRK | Cipolotti & Warrington (1996) | Eng. | NO | YES | YES | YES | YES | YES | (NR) | YES | 0.100 | (NR) |
| | | TH | Schiller et al (2001) | Eng. | YES | YES | YES | YES | YES | YES | / | YES | 0.113 | (NR) |
| | | PB | Schiller et al (2001) | Eng. | (NR) | YES | YES | YES | YES | YES§§ | / | YES | 0.215 | (NR) |

**Table 3.2: Error type effects for various patients showing primary applicability to GOB and non-GOB damage. (NR) = Not Reported. See text on following page for a description of table contents.**

* We will assume here that no mention of a semantic error in the associated reference implies no evidence for semantic errors.

† No bow-shaped curve was found here, but this was calculated on word lengths of two, three, and four letters long producing errors of 5%, 10%, and 15% respectively.

‡ The actual order of errors was DSTI, but substitutions and deletes differed by 0.8%, suggesting two qualitative interpretations of the same data.

§ From Buchwald & Rapp (2006)

** Transpositions and Inserts were both equal at 6%

†† Although FM is said to show significant imageability effects in reading, we cannot necessarily assume this is also present in spelling.

‡‡ Writing performance for function words and verbs together was significantly poorer than nouns, with function words and verbs showing no significant difference.

§§ Schiller et al state that despite PB producing slightly more errors on non-words than words, she exhibited the same monotonic increase in error rates by length for non-words as for words, suggesting that the nature of the spelling deficit was not influenced by lexical status *per se*. They would therefore categorise this as NO lexical effect.

The columns in Table 3.2 list the patient's language (**Lang.**), evidence of phonologically plausible errors (**PPE**), whether there are differences in errors across grammatical class (**Gramm. Class**), whether there is evidence of semantic errors (**Sem. Error**), whether there are more errors on abstract than concrete words (**Imageab.**), whether there are more errors on low than high frequency words (**Freq.**), whether there are more errors on non-words than words (**Lexic.**), and whether errors tend to increase as function of word length. The table also shows an *indicative* serial position curve, with a '$\Lambda$' suggesting that the patient produced a classic bow-shaped curve, and a '/' suggesting that the serial position curve increased as a function of word length. An examination of the criteria for nominating a curve as bow-shaped, monotonically increasing, or a broader range of shapes, can be found in Appendix B, but for the purposes of this chapter, we will categorise our curves as either '$\Lambda$' or '/'. A simple indication of single-error distribution is also shown by listing the first letter of each error type (**S**ubstitutions, **T**ranspositions, **D**eletes, and **I**nserts) in decreasing frequency. Thus a patient showing single errors comprised mostly of **D**eletes, followed by decreasing **I**nserts, **T**ranspositions, and **S**ubstitutions would have an error distribution of DITS. The final column to the left of the error distribution column (***m***) shows a gradient measurement based on a trend line of the word-length curve where available. We expand on this later in this chapter when we investigate a possible relationship between the gradient and the locus of impairment.

Returning to the patient groupings, our two major groups collate those patients whose symptoms suggest damage *primarily* within the graphemic buffer (group GOB), and those whose symptoms suggest damage *prior to* the graphemic buffer (group NON-GOB) within the lexical-semantic pathway (route I and IIb in Figure 3.1). The first group should therefore show bow-shaped serial position curves and a word length effect, both held to be key attributes of graphemic buffer disorder (e.g. patients SE, AS, and

LB). The presence of any semantic errors for example, or evidence of concreteness would suggest damage prior to the GOB, and such patients would therefore be placed in the second group. The second group is further subdivided into two groups. The sub-group SEM lists patients who show evidence of semantic errors (e.g. patients DA and TH). This is important as it suggests damage to the lexical-orthographic path (route IIb from Figure 3.1) and is a precondition of deep dysgraphia. The sub-group NON-SEM lists all other patients who show symptoms indicative of damage prior to the GOB, but exhibit no semantic errors (e.g. patients JH and HE). Interestingly, the NON-SEM patients also show almost no evidence of an effect of grammatical class.

In summary therefore, we do not classify these patients as DD or GBD patients. Rather, we group them according to their symptoms. Kay & Hanley (1994) for example described JH as a GBD patient, despite JH showing effects of lexicality and frequency. In addition, JH is not noted as having produced a bow-shaped serial position curve. The key motivation behind grouping patients according to symptomatic similarities is therefore to determine whether symptoms common to DD and GBD align with the predicted behaviour of Cipolotti et al's type A and type B characteristics. Results in the literature unfortunately, are usually provided in terms of single errors, and at least two key type B attribute rely on evaluating all errors: deletes, and the existence of fragments. A robust corroboration of Cipolotti et al's claims is therefore difficult. What is however clear from at least the nature of the serial position curve is that there does seem to be a relationship between symptoms of DD, and a monotonically increasing serial position curve. We now examine specific symptoms in more detail.

### 3.6.1 Concreteness, grammatical class, and frequency.

Patients suffering from DD are expected to show an effect of concreteness and frequency such that more errors are made on abstract (low imageability) words than

concrete (high imageability) words. Evidence of these effects was shown by all patients listed in the NON-GOB group of Table 3.2 where data was available. Words of different grammatical class also show different error rates, with nouns generally being less erroneous than verbs. Patient DA (Cipolotti et al) for example showed better performance on concrete nouns than verbs and functors, and MRF (Orpwood & Warrington, 1995) showed significantly poorer performance on non-words than words, and on verbs compared to nouns. The effects of concreteness, frequency, and grammatical effects can also overlap, with high frequency words being better than low frequency words across concreteness and grammatical class. Bub & Kertesz (1982) for example compared abstract and concrete nouns firstly to determine the impact of abstractness on performance, and secondly because nouns are typically less susceptible to error than verbs or functors.

### 3.6.2 Word length effect

Patients with GBD show evidence of producing more errors on longer words than shorter words and is a key indicator that the GOB is affected in some way (e.g. Baxter & Warrington, 1983, Badecker et al, 1990, Caramazza & Miceli, 1990, McCloskey et al, 1994, Orpwood & Warrington, 1995, Cipolotti & Warrington, 1996, Jonsdottir et al, 1996, Schiller et al, 2001, Cipolotti et al, 2004). This effect can be more pronounced in some patients than others. Patient ML (Hillis & Caramazza, 1989) for example showed much poorer performance on six, seven, and eight letter words than four and five letter words (40% to 100%), whereas patient LB (Caramazza & Miceli, 1990) showed less difference between error rates (5% to 77%) across all word lengths (see Figure 3.2). Similar (non graphemic) behaviour has also been noted in aphasic patients with error rate being a function of the number of syllables in the word (e.g. Friederici et al, 1981).

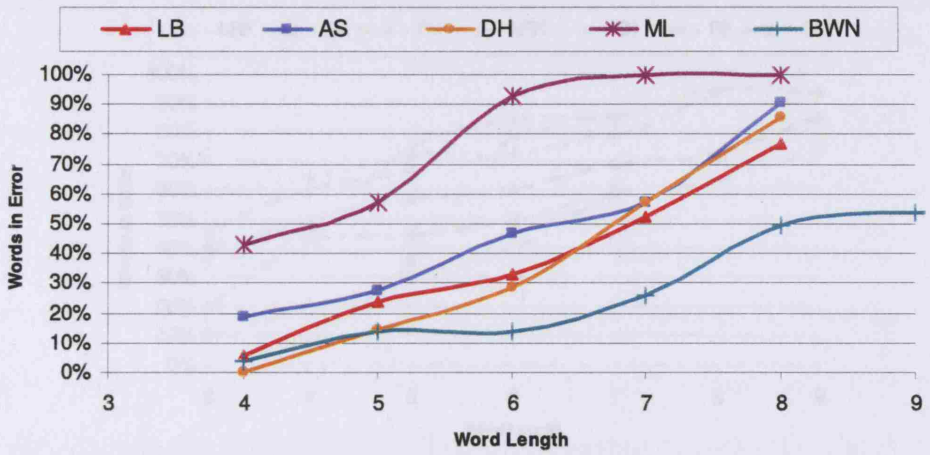



**Figure 3.2: Word length behaviour of five GOB-group patients, LB (Caramazza et al, 1987), AS (Jonsdottir et al, 1996), DH and ML (Hillis & Caramazza, 1989), BWN (Tainturier & Rapp, 2004).**

As Figure 3.3 shows, a noticeable word-length effect was also seen in SEM patients. Patient PB (Schiller et al, 2001) exhibited large differences, 7% for four letter words to 93% for eight letter words, whereas BA (Ward & Romani, 1998) showed far fewer errors for the same length words, 50% for four letter words, and 73% for eight letter words. A word length effect is considered intrinsic to the GOB indicative of a general inability of the *buffer* to cope with longer words, the general supposition being that words with more letters provide more opportunity for errors. Posteraro et al (1988) suggest that perhaps the graphemic buffer makes more errors when overloaded with too much information. Two patients, DS (Chialant et al, 2002), and OM (Miozzo et al, 2002) showed no variance in errors as a function of word length in their error behaviour, the authors citing such behaviour as evidence of no GOB damage at all.

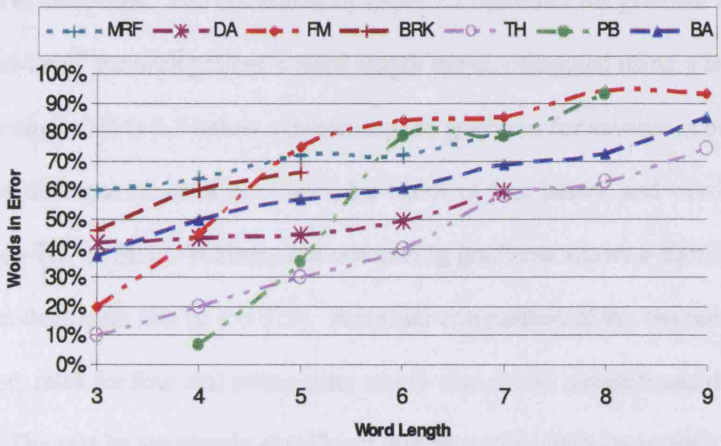



**Figure 3.3: Word length errors of seven SEM patients. MRF, DA, FM, BRK, TH, PB, and BA.**

Although both patient groups exhibit a word length effect, there appears to be a slight difference in the steepness of trend lines for word length curves between GOB patients, and NON-GOB patients. It is therefore possible that there is a relationship between damage to the GOB, and the steepness of the curve. As discussed in 3.6, patients listed in Table 3.2 were grouped according to their *predominant* symptom types. However, as we discuss later, a number of NON-GOB patients clearly showed bow-shaped error curves (FM, JES, AM, and BH), which indicate damage to the GOB. Despite damage elsewhere in the model for these patients, we would argue that the word-length effect reflects the GOB's ability to cope with serializing a buffered set of characters. Thus, performance on longer words for patients with a damaged GOB would be worse than on short words.

### 3.6.2.1 A predictable word-length effect

Table 3.2 shows that where data is available, all patients show a consistent word length effect; longer words show more errors than shorter words, and such an effect is usually considered indicative of damage to the GOB. Although both GOB and NON-GOB patients produce a word length effect, we believe they are qualitatively different in terms

of their relative steepness. The column ***m*** in Table 3.2 describes the gradient associated with the trend-line[10] for each patient's word length curve, computed using a least-squares algorithm. Table 3.3 below summarises the gradients for seventeen patients and indicates how different the error rates were for words of four letters, and words of seven letters (**Diff (4-7)**). A Mann-Whitney test comparing gradients shows a significant difference between both sets ($p < 0.015$). A similar comparison of the overall difference between errors rates for four and seven letter words also shows a significant difference ($p < 0.011$). The results are clearly significant, but seventeen patients is not a large data set, and we believe that further evidence and patient results could only serve to validate this further. Additional work may show that damage in the GOB consistently produces significantly steeper word length curves. A shallow curve for example, may indicate that despite a word length effect, the GOB itself is functional, simply producing errors as a result of degraded input. In other words, a truly damaged GOB would produce far more errors as a function of word length. We discuss this further in Chapter 8, but the simulation model described in Chapter 5 produces similar results suggesting that the steepness of the word length curve may indeed relate to the locus of impairment.

[10] The *m* term comes from equation $y=mx + c$, where m represents the gradient or steepness of the trend

| Putative Evidence of GOB damage | Gradient | Diff. (4-7) | Putative evidence of no GOB Damage[11] | Gradient | Diff. (4-7) |
|---|---|---|---|---|---|
| LB | **0.171** | 0.463 | MRF | **0.048** | **0.160** |
| AS | **0.174** | 0.385 | DA | **0.042** | **0.162** |
| DH | **0.214** | 0.571 | BRK[12] | **0.100** | **0.060** |
| ML | **0.157** | 0.571 | TH | **0.109** | **0.380** |
| BWN | **0.093** | 0.216 | **PB** | **0.214** | **0.429** |
| AM[13] | **0.139** | 0.417 | BA | **0.051** | **0.191** |
| JES | **0.129** | 0.500 | JH | **0.043** | **0.250** |
| BH | **0.194** | 0.636 | HE | **0.114** | **0.429** |
| FM | **0.117** | 0.400 | | | |
| **N=9, Average** | **0.154** | **0.462** | **N=8, Average** | **0.090** | **0.258** |

**Table 3.3: Comparative gradient values for word length curves for GOB and non-GOB damage**

We cannot provide a strong argument for the type of performance produced by PB (Schiller et al, 2001, highlighted in Yellow in Table 3.3), namely the presence of a high gradient and large error range. Although there is a word-length effect, the absence of a bow-shaped serial position curve suggests no damage to the serial production mechanism itself. Errors by PB tend to affect letters after the first few serial positions (e.g. ODOR → ORDER, READY → REACY). Since a general tendency to produce the correct letters for early positions in words is also common to patient BA (Ward & Romani, 1996), a logical question arises as to why BA did not show a comparable lack of errors associated with shorter words? BA's results were calculated on a corpus of 2000 words, which were not spread out evenly across lengths of three to eleven letters. Patient PB's errors were calculated on 90 words, where each word length, (four to eight) had fourteen words. In short, PB's anomalous result may simply be due to a lack of sufficient data.

### 3.6.3 Error distributions

Damage to the GOB could result in loss of letter identity and serial order, so patients are expected to make substitutions, inserts, transpositions and deletes. The distribution of

[11] Damage elsewhere suggests that the GOB appears to be intact.
[12] Only word lengths 4 and 5 were used here
[13] Word lengths used were short (3-5 letters), and long (6-8 letters)

these errors is not consistent as can be seen from the relative proportions of single errors in Figure 3.4. SE for example produces mostly deletes, whereas FV produces mostly substitutions (65%), and far fewer inserts (21%), deletes (11%), and transpositions (3%)

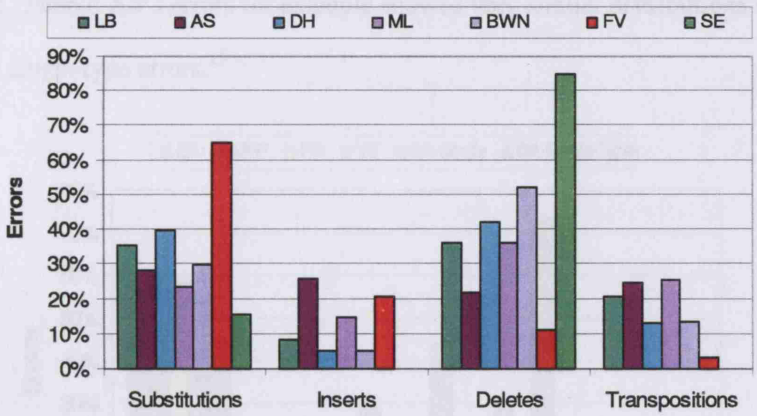



**Figure 3.4 : Error Distributions for single errors for various GOB patients**

Although distributions for the NON-GOB (SEM) patients in Figure 3.5 are also inconsistent, there seems to be less variance than for GOB patients. Both patient groups produce a majority of substitutions and deletes, and GOB patients produce the fewest inserts. Figure 3.5 shows single errors of NON-GOB semantic patients. For patient BA, we show single as well as *single-type*[14] errors to compare their relative distributions. Ward & Romani (1998) originally investigated a phenomenon typical of BA's error behaviour, namely the presence of *fragments*; an erroneous word whose length is two or more letters shorter than the target and thus cannot count as a single error. In presenting BA's errors against other NON-GOB patients, we therefore provide two analyses of BA's errors. The first summarises single errors (hence omitting fragments), and the second summarises single-type errors, which does indeed show a propensity to produce numerous deletes (and fragments). Excluding BA's single-type errors, NON-GOB (SEM) patients do seem to show less variance in error distributions than GOB patients.

[14] See Caramazza et al (1987), and Caramazza & Miceli (1990) for a comparison.

This may also suggest that the GOB copes more consistently with degraded input, than it can do when damaged. In addition, it seems that single errors by themselves may be an inadequate measure of the effect of damage, at least as far as type B patients are concerned. Patient AS's errors for example showed very similar distributions for single errors and single-type errors.[15]

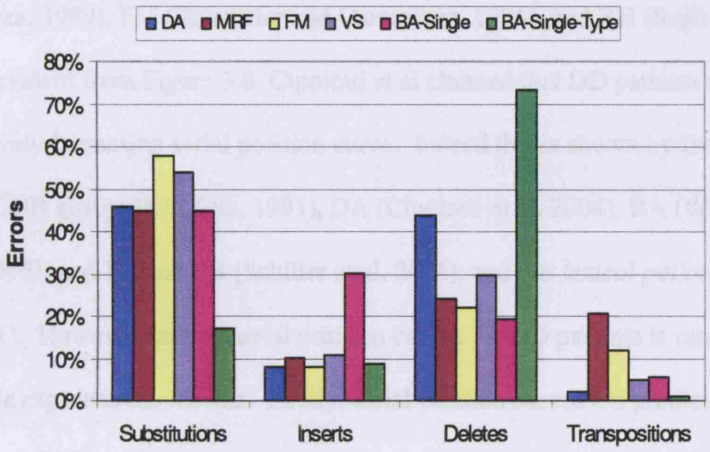



**Figure 3.5 : Error Distributions for single errors for various NON-GOB(SEM) patients, and single-type errors for patient BA.**

Contrary to Cipolotti et al's type A and B categories, Table 3.2 shows five out of seven GBD patients with a majority of deletes, and four out of five NON-GOB semantic patient distributions showing a majority of substitutions. We remind the reader that Cipolotti et al do not define their error distributions in terms of *single* errors, and so it is difficult to substantiate whether patients in Table 3.2 do indeed conform to their characteristic type A and type B behaviours.

Our next investigation examines various patients classified according to the type of serial position curve they produced, the rationale behind this being that according to Cipolotti et al, a bow-shaped error curve suggests damage to the GOB, whereas a monotonically increasing error curve suggests degraded input to the GOB.

[15] Single errors were 28.5%, 25.6%, 21.6%, and 24.6%, and single type-errors were 34.6%, 26.5%, 17.7%, and 21.2% for substitutions, inserts, transpositions, and deletes respectively.

### 3.6.4 Serial position effects

Both GOB and NON-GOB patients show an effect of serial position in the sense that errors tend to produce a predictable pattern. Damage to the GOB is expected to produce a bow-shaped curve with errors more likely in medial positions than at either end. This was shown by LB (Posteraro et al, 1988), AS (Jonsdottir et al, 1996), ML and DH (Hillis & Caramazza, 1989), FM (Tainturier and Caramazza, 1996), and BH (Sage & Ellis, 2004) and evident from Figure 3.6. Cipolotti et al claimed that DD patients should exhibit a progressively increasing serial position curve. Indeed this is shown by five patients in our NON-GOB group; HR (Katz, 1991), DA (Cipolotti et al, 2004), BA (Ward & Romani, 1996), and TH and PB (Schiller et al, 2001), and our *lexical pathway* patient HR (Katz, 1991). However data on serial position curves for DD patients is rare. There are two possible explanations for this. Firstly, serial position curves are predicted to be due to suboptimal GOB performance, and usually analysed for graphemic buffer patients. Secondly, DD patients usually have their behaviour analysed in the context of semantic and syntactic dimensions such as imageability and word-class, thus not concentrating on symptoms associated with damage to the GOB.

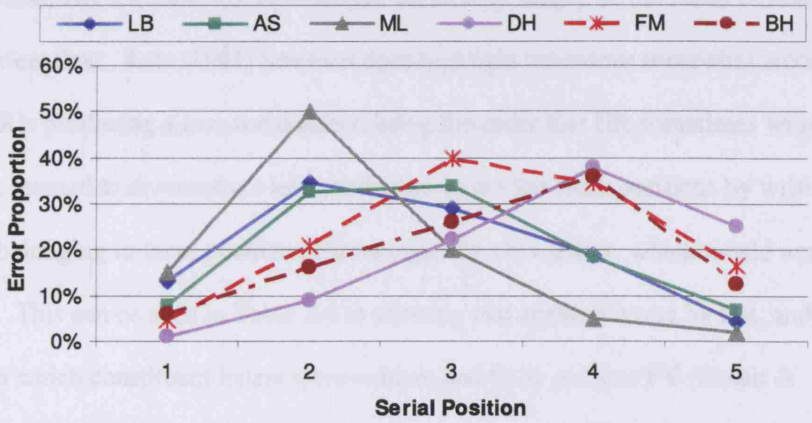



**Figure 3.6: Normalised single error positions for six patients exhibiting damage to the GOB – LB (Caramazza & Miceli, 1990), AS (Jonsdottir et al, 1996), ML and DH, (Hillis & Caramazza, 1989), FM (Tanturier & Rapp, 2004), and BH (Sage & Ellis, 2004).**

In contrast to the bow-shaped curve shown for GOB patients, NON-GOB patients show a different type of serial position curve with errors tending to increase monotonically as a function of letter position (Figure 3.7)[16]. One of the first recorded patients to demonstrate this behaviour was HR (Katz, 1991) who does indeed show a qualitative propensity to produce serially increasing errors despite producing a strictly bow-shaped curve.

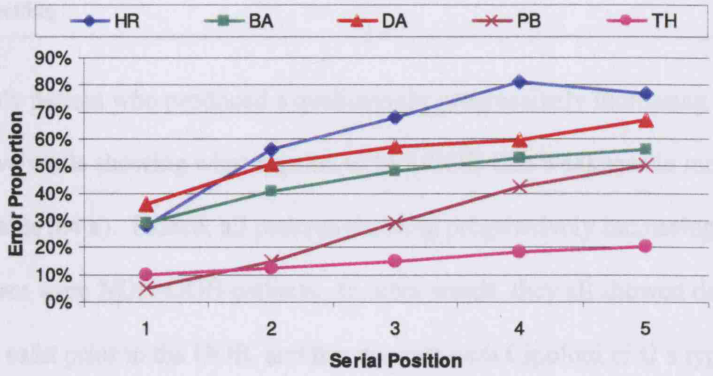



**Figure 3.7: Normalised serial position errors for five patients with type B serial curves HR (Katz, 1991), BA (Ward & Romani, 1996), DA (Cipolotti et al, 2004), PB & TH (Schiller et al, 2001).**

[16] A Mann-Whitney comparison between the highest serial positions associated with patients in Figure 3.6 and Figure 3.7 showed a statistically difference ($p < 0.008$).

In isolation, HR's technically bow-shaped curve may simply be the result of noisy or insufficient data. Katz (1991) however does highlight behaviour somewhat incongruous with HR's producing a true serial effect, being the order that HR sometimes wrote letters in. HR seemed to demonstrate less confidence in medial letter positions by writing letters belonging to these positions chronologically after letters, which would normally follow. This can be seen in Table 3.4 a) showing two spelling errors by HR, and the order in which constituent letters were written, and b) by patients FV (Nolan & Caramazza, 1985) and AM (de Partz, 1995) for correctly spelled words.

| | | | |
|---|---|---|---|
| a) | PROVE → P O O V E | | 1 2 5 3 4<br>MOVE → M I V E<br>1 2 4 3 |
| b) | MERCANTILE | → | 1 2 3 4 8 9 10 5 6 7<br>M E R C A N T I L E |
| c) | CADAVRE | → | 1 2 6 5 3 4 7<br>C A D A V R E |
| | PAROLE | → | 1 4 5 6 2 3<br>P A R O L E |
| | BRINDILLE | → | 1 2 6 7 8 9 3 4 5<br>B R I N D I L L E |

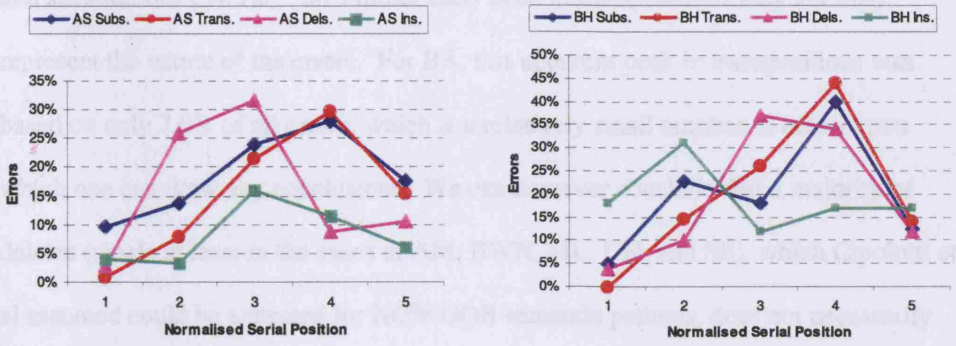
**Table 3.4 : a) Sample errors by HR (Katz, 1991), b) a correctly written word by FV (Nolan & Caramazza, 1983), and c) correctly written words by AM (De Partz, 1995) indicating written letter order when spelling**

HR is the only patient who produced a qualitatively progressively increasing serial position curve while showing what appears to be a GOB-like weakness in medial serial positions (Table 3.4 a). Indeed, all patients showing progressively increasing serial position curves were NON-GOB patients. In other words, they all showed damage presumed to exist prior to the GOB, and this concurs with Cipolotti et al's type B characterisation. Although there are four NON-GOB patients who show a bow-shaped serial position curve, we believe this indicates multiple loci of impairment with damage in the GOB as well as the lexical route. An example of non-graphemic bow-shaped error

curves based on syllable position behaviour has also been noted in aphasic patients (Friederici et al, 1981) with errors being more prominent in medial syllables than at either end. The results were averaged over 12 aphasic patients of whom eight had Broca's aphasia, which may have involved damage to both the semantic route as well as the GOB.

## 3.7 Error Type across Serial Position

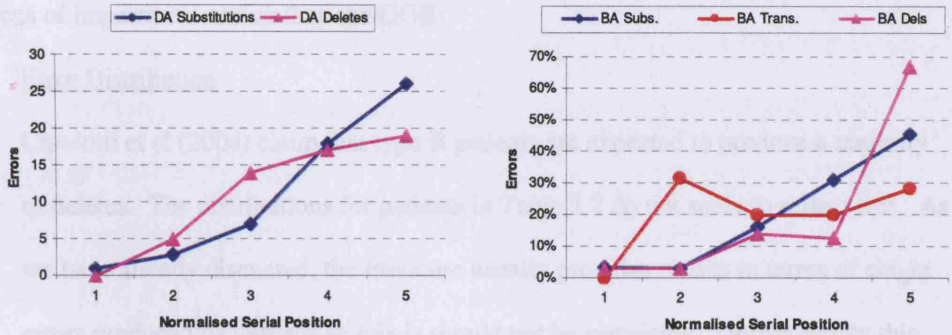
The literature describes the overall behaviour of errors across serial position, yet there is less discussion of how constituent error types behave across those same serial positions. Are deletes for example, more prevalent towards the beginning or end of words? Do transpositions occur in medial positions, or across the entire word length? Figure 3.8 shows constituent error curves for AS and BH who both produce an overall bow-shaped error curve. Substitutions, transpositions and deletes all produced qualitative bow-shaped curves peaking in serial positions four, four and three respectively, with the major difference being that AS produced more deletes earlier in the word than BH. Inserts seemed to show very different behaviour across serial positions for both patients, so it is difficult to generalise what *typical* behaviour would be.




**a) – Patient AS** **b) – Patient BH**

**Figure 3.8: Constituent error types for two patients producing an overall bow-shaped serial curve**

Figure 3.9 shows constituent error curves for DA and BA who both produce an overall increasing serial position curve. Unfortunately, these were the only NON-GOB patients where constituent serial position data could be found, making it difficult to draw robust conclusions about patterns of similar activity. Deletes and substitutions for both patients showed the serially increasing curve described by Cipolotti et al as being typical of type B patients.




**a) – Patient DA** **b) – Patient BA**

**Figure 3.9: Constituent single error types for two patients returning a serially increasing error curve.**

Transpositions for patient BA on the other hand showed a much less convincing pattern. The number of transpositions (7.9%) was relatively minor compared to deletes (27.5%) and substitutions (64.5%). So a minor early peak in transpositions may not truly represent the nature of the errors. For BA, this apparent peak in transpositions was based on only 7.9% of all errors, which is a relatively small number of errors upon which one can draw any conclusions. We can however conclude that a majority of deletes (single at least in the cases of AM, BWN, ML, DH, and SE), which Cipolotti et al assumed could be expected for NON-GOB semantic patients, does not necessarily account for an increasing serial positing curve. Many patients showing such behaviour produced bow-shaped serial position curves.

## 3.8 Common Origins of GBD symptoms

Reviewing Table 3.2, it is clear that GOB and NON-GOB patients show distinct differences in symptoms related to grammatical class, concreteness, frequency and lexicality. There seems to be almost no difference between the GOB patients, whereas this is clearly more pronounced for the NON-GOB patients. Let us consider GBD symptoms shown by DD patients, and how each might be associated with a presumed locus of impairment earlier than the GOB:

1. Error Distribution

   Cipolotti et al (2004) claim that type B patients are expected to produce a majority of deletes. The distributions for patients in Table 3.2 do not seem to reflect this. As we have already discussed, the literature usually provides results in terms of single errors produced by patients so this is should not be surprising. Unfortunately this also means that patients who tend to produce fragments, a key type B characteristic, are also not highlighted.

2. Word Length Effect

   Table 3.2 which summarises the symptoms produced by each patient shows a word length effect for all of them (except FV) irrespective of group. Data from seventeen patients suggested that locus of impairment may indeed affect the steepness of the word length curve. Clearly access to substantially more data from patients showing evidence of *pure* GBD, and DD may be able to provide more conclusive evidence that the word-length gradient differs as a function of locus of impairment.

3. Serial Position Effect

   There is also clearly a difference in serial curves for GOB patients who produce the classic bow-shape, and NON-GOB patients who produce what at a superficial level might appear to be right-side neglect errors. The idea that the GOB itself may be

selectively damaged has been suggested by Cubelli (1991) referring to an inability to produce vowels, so selective damage to an underlying mechanism affecting the type of serial curve may also be possible. We discuss serial mechanisms in more detail in Chapters 5 and 6 and show that selectively lesioning a connectionist model can produce both bow-shaped and monotonically increasing serial position curves.

## 3.9 Geminate Behaviour

Errors with double letters are thought to originate in the GOB. A number of theories attempt to explain why patients produce these errors, and patient data is not always consistent. Patient LB (Caramazza & Miceli, 1990) for example, performed better on six-letter geminate words than six-letter non geminate words. For example, spelling for words such as STELLA was better than words such as SCONTO. Caramazza & Miceli argued that according to the word length effect, six-letter words with geminates actually behaved like five-letter words (with an active geminated letter) and thus are expected to be less erroneous than six letter words (with no geminates). In DA's (Cipolotti et al, 2004) case, 25 geminate words of lengths four, five, and six were frequency matched to 25 non-geminate words. Words with geminates were much less prone to errors. Patient HE (McCloskey et al, 1994) however, appeared to have particular difficulty spelling words containing geminates. HE misspelled 26% of geminate words, but only 13% of non geminate words, with most spelling errors in geminate words implicating the geminate (e.g. CROSS → CROOS, and JEEP → JEP). Matching for word length was not clearly applied in HE's case. Patient AS (Jonsdottir et al, 1996) showed comparable results on both word types with 18.7% of geminate and 19.1% of non geminate words in error. Although we will discuss the nature of geminates as they relate to our model in

substantial detail in Chapter 7, we now briefly introduce the general behaviour of doubling errors with respect to GBD patients.

Evidence suggests that consonant/vowel status may be represented by a separate *tier* during spelling (e.g. Caramazza & Miceli, 1990, Miceli et al, 2004, Tainturier & Caramazza, 1996, McCloskey et al, 1994). Caramazza & Miceli (1990) proposed that geminates have a distinct identity with orthographic representations consisting of at least a consonant/vowel status tier and a grapheme identity tier (see also McCloskey et al, 1994 for their *Multiple Tier Hypothesis*). These tiers in Figure 3.10 (a) for the word BASKET and in Figure 3.10 (b) for the word RABBIT shows the proposed doubling feature.


**Figure 3.10: (a) Representation of grapheme identity and consonant/vowel status, and (b) Representation of a doubling status as per Caramazza & Miceli (1990)**

In reviewing word-length comparisons by Caramazza et al for LB, errors on six-letter words containing a single geminates would therefore be compared to five-letter words without geminates since the multi-tier model suggests a comparison at the graphemic-identity tier alone. Evidence also exists to support the view that geminated letters do not produce errors in the same way as single letters (e.g. Caramazza & Miceli, 1990, McCloskey et al, 1994, 1996, Tainturier & Caramazza, 1996). Patients do not all provide consistent behaviour, yet will generally show a combination of any/all of the following symptoms:

1) The letter identity may be impaired, with geminate information preserved leading to another letter being doubled in place of the original, for example, PARROT →

PATTOR by patient FM (Tainturier & Caramazza, 1996). Note also the transposition.

2) The geminate status may *move* elsewhere in the word, for example, BREEZE → BREZZE by patient AS (Jonsdottir et al, 1996).
3) The geminate status can be lost. E.g. BOCCA → BOCA by patient OM (Miozzo and De Bastiani, 2002). Badecker (1996) has also termed this *geminate shortening*.
4) Words containing no doubles rarely show doubling errors. In delayed copy transcoding tests for example, FM only generated additions in words already containing double letters. Some exceptions by FM were explained as being due to a two-step process involving first a semantic error followed by a graphemic buffer error on the new word. One instance argued by the authors is the response ELEPHANT → ROTIGGE as follows: ELEPHANT → GIRAFFE → ROTIGGE. Note the consistency in geminate position and adherence to consonant/vowel status. BA[17] showed what we would argue is a comparable example - PUDDING → DESERT → DESSET. In BA's case however, the geminate status appears to have persisted affecting the paraphasia. One notable exception to the expected behaviour is demonstrated by patient AS, producing almost 50% of geminate related errors on words without doubles. This is discussed further in Chapter 7.

## 3.10 Conclusion

In this chapter we described the core symptoms associated with GOB and NON-GOB patients identifying some common properties. These are generally described in terms of errors associated with concreteness, grammatical class, frequency, error distribution word-length and serial position. The existence of such symptoms is usually indicative

[17] From BA's raw data provided by Jamie Ward.

of where damage is situated in the spelling model, yet using dissociation to isolate the specific locus of impairment is rarely a simple process (e.g. Shallice, 1988). Cipolotti et al describe two putative function syndromes, type A and type B GBD, which they claim arise as consequence of GBD and DD related damage respectively. Their proposed type A and type B GBD classifications provide an insight into the general symptoms one might expect to be produced by GBD and DD patients respectively. However, subjecting their classification to a broader range of patients shows that their originally claimed properties may need refinement. Firstly, the absence of substantial data showing *total* errors makes the current classifications difficult to corroborate. Secondly, at least in terms of single errors, GBD patients who one would expect to exhibit type A behaviour also show type B behaviour in the sense that they produce a majority of deletes, and a majority of deletes may also produce many fragments. Thirdly, the existence of patients such as JES, AM, BH, and FM show that damage to the lexical pathway cannot confidently predict type B serial position behaviour. It is true that these patients seem to have damage in the GOB as well, and this therefore highlights another possible issue with Cipolotti et al's classification. Namely, that patients would require relatively pure disorders to correspond to their classification.

One objective of this chapter was to investigate further whether Cipolotti et al's type A and type B classifications matched the behaviour associated with a broader range of patients than they initially investigated. As we have discussed, this has proved difficult without access to more comprehensive patient data. Clearly, we cannot claim that the Cipolotti et al classification is flawed since their study does indeed have access to their patient data. One characteristic which generally encompasses all errors when published is the serial position curve, which seems to behave as they predict. We would therefore agree that their categorisations broadly indicate a putative functional

relationship between damage presumed to occur prior to the GOB, and apparent suboptimal performance within the GOB as the result of a cascaded, or downstream effect of that prior damage. Our comparison of the steepness of the gradient curves produced by patients with damage presumed to be located in, and prior to the GOB shows further evidence that a functional relationship between the behaviour of the GOB, and patient errors may exist. It may also provide evidence for the addition of another characteristic in future type A and type B classifications.

The rest of this thesis develops further a connectionist model originally put forward by Glasspool et al (2006), and as we will clarify in more detail throughout the thesis, the loci of impairment in Glasspool et al's original model are theoretically linked to Cipolotti et al's type A and B behaviour. As discussed previously, Cipolotti et al's claims rely on the analysis of total errors, and we would expect that opportunities to investigate their claims in more detail are likely to arise as more comprehensive patient data is published. Therefore without evidence to the contrary, we will use Cipolotti et al's type A and type B classifications to refer to patients who are presumed to show damage within, and prior to the GOB respectively.

In the next chapter, we will discuss types of neurological damage found in the presence of acquired disorders and illnesses. We will then draw some connectionist parallels used to apply similar damage to models of aspects of neurological processing.

# 4 Lesioning Connectionist Systems

## *4.1 Introduction*

The field of connectionism provides many examples of damage to supposedly brain like structure in artificial neural networks and actual disorder in physical nervous systems (e.g. Hinton & Shallice, 1991, Houghton et al, 1994, Plaut et al, 1996, Horn et al, 1996, Mozer et al, 1997, Camperi & Wang, 1998, Braver et al, 1999, Glasspool et al, 2006). Many of these simulate the effects of stroke, tumours or progressive disorders such as Alzheimer's. The Motivation for using connectionist models in modelling cognitive behaviour can be quite compelling as connectionism is a field inspired by real neuronal structures.

Moving from theory to practice however, is not always straight forward. O'Reilly (1998) for example, claims that it can be difficult to apply biological principles. Known biology often provides insufficient constraints, and focusing on realism often reduces to plausibility arguments on facets of design that are not inconsistent with known biology. A good example of where this critique is appropriate is the back propagation algorithm (e.g. Rumelhart, Hinton, & Williams, 1986). O'Reilly describes six principles for computational cognitive neuroscience models: 1) biological realism, 2) distributed representations, 3) inhibitory competition, 4) bi-directional activation propagation (interactivity), 5) error-driven task learning, and 6) Hebbian model learning. Of these, he claims that only distributed representations, and error-driven task learning are shown by backpropagation. A more positive view has been put forwarded by Cohen & Servan-Schreiber (1992: 53) who argue that "*like statistical mechanical models in physics and chemistry, connectionist models are designed to capture those features of a lower level system (information-processing mechanisms in the brain) that are most*

*relevant at a higher level of analysis (cognition and behaviour). Thus, an important goal in constructing such models is to make it possible to examine the effects of biological variables on behaviour without having to reproduce the entire brain*". They suggest further that backpropagation implements the general phenomenon of gradient descent and that gradient descent has been a powerful concept for describing many of the details concerning human learning behaviour. Backpropagation they argue offers a reasonable approximation to the *type* of learning that occurs in neural systems, even if the actual algorithm is different. We would agree whole heartedly with Cohen et al's view but O'Reilly's provides a sound reminder that neural networks are crude approximations to nervous system structures and that behavioural realism found in connectionist models cannot then simply be used to make claims about neural structure.

Not all popular network types are as controversial in their plausibility as backpropagation. Self-organising networks (Kohonen, 1997) for example, use an unsupervised algorithm such that individual units learn to specialise on similar patterns, thus becoming feature detectors. This behaviour has been seen in topographic maps in the visual system, where neurons lying along a line or column orthogonal to the surface of the primary visual cortex (V1) respond in approximately the same way to visual stimuli (e.g. Sincich & Blasdel, 2001)

One apparent benefit of inflicting pathological damage to these types of models is that they can be controlled precisely, and systematically modified over arbitrarily large numbers of experimental *quasi-subjects* and information processing tasks. As Ruppin (1995) claims, these experiments are open to detailed inspection in ways that biological systems are not. These may be inexact models of neurological behaviour, yet some authors have attempted to quantify the activity of low-level neuronal functionality using concise mathematical formulae, which can then be used as candidate functions in

neural network models of the nervous system. Dayan & Abbott (2001) for example, provide in-depth discussions of many formulae, which attempt to explain many characteristics of neural activity using various connectionist models, and across a broad range of neuron types. These include the simulation of neuronal firing rates, white noise stimuli, intracellular resistance and membrane capacitance. As we will discuss further, many authors have applied such principles to a variety of connectionist models using feed forward, recurrent, and excitatory-inhibitory networks. Models of spelling production for example represent higher level functionality, and the realism of such models is open to debate. However, investigation at this level of detail seems necessary if analysis using connectionism is to achieve a level of credibility. It is also possible to investigate the effects on models in ways one might practically and ethically be unable to with human subjects.

If connectionist systems can be used to model normal behaviour due to a level of similarity to the nervous system, then we might also consider damaging connectionist networks in ways that nervous systems are damaged. Small (1994) describes three ways in which damage or lesioning can occur: 1) focal lesions representing damage to well circumscribed regions of the brain or computer model, for example stroke; 2) diffuse lesions involving damage to a large number of neural elements over a widespread area of the brain or model, as for example in Alzheimer's disease, and 3) patch lesions consisting of multiple small areas of circumscribed damage, such as occurs within the temporal lobe and limbic system from herpes simplex, encephalitis and paraneoplastic limbic encephalitis.

## *4.2 Approaches to Lesioning Connectionist systems*

If real nervous system components and their interactivity have inspired artificial neural networks, then the nature of physiological damage may also provided similar inspiration

for techniques used for artificial damage. We outline six methods of inflicting damage to connectionist systems and provide crude mathematical approximations to how these might impede the natural workings of intact models. These are simplifying approximations and we make no claims that they accurately reflect the real mechanics of the nervous system. They do however provide us with a methodical approach to inflicting damage to our models in ways *inspired* by similar damage to the nervous system. In a similar vein to the Cohen & Servan-Schreiber's (1992) comment regarding backpropagation, we believe our techniques offer a "reasonable approximation to the *type* of damage that occurs in neural systems, even if the actual algorithm is different". These lesion types can be applied in a *focal* or *diffuse* manner. An example of focal lesioning in a feedforward network would involve removing specific subsets of connections (e.g. Hinton and Shallice, 1991, Plaut, 1995) and/or hidden units (e.g. Medler, Dawson, & Kingstone, 2005), or even modifying activation functions in a predictable manner (e.g. Cohen & Servan-Schreiber, 1992, Usher & Davelaar, 2002, Camperi & Wang, 1998). An example of diffuse lesioning in a connectionist system may involve scaling (Houghton & Zorzi, 2003, Glasspool et al, 2006), or adding noise to all connection weights (e.g. Bullinaria 2004).

The six lesion techniques we apply in our model are 1) Ablation, 2) Noise, 3) Scaling connection weights, 4) Constraining connection weights, 5) Modifying an activation function, 6) Adding noise to an activation function. We will describe each of these in more detail further in the chapter. Table 4.1 lists a number of connectionist models where many of these approaches have been used to simulate various aspects of cognitive behaviour, each applying one or more of these lesion types.

| Domain of simulation | Lesion approaches | Reference |
|---|---|---|
| Aspects of deep dyslexia, and semantic access dyslexia. | Ablation, Noise | Hinton and Shallice (1991) |
| Verbal short term memory | Noise on activation function | Burgess & Hitch (1992) |
| Connectionist model of the Stroop effect | Modification of activation function. | Cohen & Servan-Schreiber (1992) |
| Optic Aphasia | Ablation | Plaut & Shallice (1993) |
| Serial order errors in Graphemic Buffer Disorder | Noise on activation function | Houghton, Glasspool & Shallice (1994) |
| Double dissociations related to modularity. | Ablation | Plaut (1995) |
| Surface Dyslexia | Ablation, Noise, Unit Removal | Plaut, McClelland, Seidenberg, Patterson (1996) |
| Memory deterioration in the presence of Alzheimer's | Ablation, Scaling | Horn, Levy & Ruppin (1996) |
| Deep dysgraphia and semantic dementia | Ablation, Noise | Wright & Ahmad (1997) |
| Lexical access in aphasia | Noise, Scaling | Dell, G.S., Schwartz, M.F., Martin, N., Saffran (1997) |
| Unilateral Neglect | Noise | Mozer, Halligan & Marshall (1997) |
| Semantic impairments in the presence of Alzheimer's | Ablation | Devlin, Gonnerman, Andersen, Seidenberg (1998) |
| Visuospatial working memory | Modification of activation function, Noise | Camperi & Wang (1998) |
| Past tense processing | Ablation | Joanisse & Seidenberg (1999) |
| Schizophrenia – models of the effect of dopamine on behaviour | Noise, Activation Noise. | Braver, Barch & Cohen (1999) |
| Modelling attentional neuromodulation | Modification of activation function | Usher & Davelaar (2002) |
| Separable processing of consonants and vowels | Noise | Monaghan & Shillcock (2003) |
| Surface Dysgraphia | Scaling | Houghton & Zorzi (2003) |
| Modelling symptoms of Graphemic Buffer Disorder and Deep Dysgraphia | Activation Noise, Scaling | Glasspool et al (2006). |

**Table 4.1: Various connectionist networks applying artificial lesioning.**

This is by no means an exhaustive list of models using such lesion types. It does however demonstrate the broad range of applications possible using connectionist modelling and their associated artificial damage. There are other lesion types not described in this thesis. Kohonen self-organising maps for example, may be lesioned by removing *winning* output nodes, thus simulating loss of cell matter. Our investigations

however, only review methods applied in our model, which uses feed forward multi-layer networks with non-linear activation functions.

Throughout the thesis, we damage our model using various severities, at various locations using one of six lesion types described further in the rest of this chapter. The model we damage is described in more detail in Chapters 5, 6, and 7. However, we will be precise about the location of the damage, for example "all **connections** leading from **hidden nodes** to the **geminate node**". Lesion severity refers to the extent of damage applied to the network and will change depending on the specific lesion type being applied. This may take the form of disturbance to an individual connection (focal), or damage to multiple connections (diffuse). Unlike some authors, we lesion our networks in a relatively simple fashion. A more sophisticated technique used by Ruppin (1995) for example, applied spatial lesions to his network in order to investigate whether slit or round forms affected overall network performance differently.

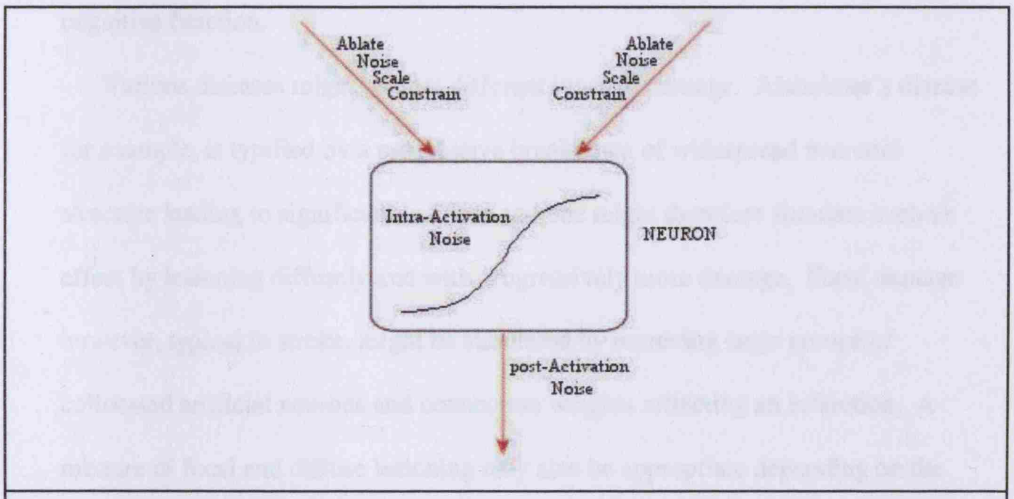



**Figure 4.1 : A simple view on how artificial lesions are applied to an artificial neuron in the thesis.**

We now discuss our lesion types in more detail providing biological parallels where possible. All lesion types can be simulated in a diffuse or focal manner. However, we will apply all of them diffusely unless otherwise specified. Figure 4.1 shows in simple

terms how each technique can be applied to the basic artificial neuron used in the thesis. Each of these can be lesioned to varying degrees, and a summary of the values used to simulate each of 10 progressively increasing severities can be found in Appendix D.

1) **Ablation**

Ablation is characterised by an effective loss of the neuron or connecting tissue, and may be caused by trauma, stroke, or disease. Stroke can be of two types, ischemic or haemorrhagic. Ischemia, characterised by a loss of circulation to an area of the brain as a result of blockage, results in infarction and a loss of neurological function. The effect of such an event is usually immediately obvious and accompanied by severe cognitive dysfunction. Haemorrhage is characterised by bleeding into neural tissue. Subsequent clotting may put pressure on nearby brain tissue restricting the delivery of nutrients and oxygen. Unlike the relatively rapid effects seen as a result of trauma or stroke, disease is usually manifested by a gradual degradation of neural tissue and cognitive function.

Various diseases might present different types of damage. Alzheimer's disease for example, is typified by a progressive breakdown of widespread neuronal structure leading to significant cell loss and one might therefore simulate such an effect by lesioning diffusely and with progressively more damage. Focal damage however, typical in stroke, might be simulated by removing large groups of collocated artificial neurons and connection weights reflecting an infarction. A mixture of focal and diffuse lesioning may also be appropriate depending on the disorder being simulated. The effects of hydrocephalus for example, can be seen at a diffuse level through excessive levels of cerebrospinal fluid resulting in a disproportional pressure on the brain. There may also be localised loss of function suggesting focal lesioning.

Ablation is the most intuitive of lesion approaches, and reflects the complete breakdown of an axon, or ability of a neuron to participate in cognitive operations. We simulate this by setting the weight between connected nodes to zero. The severity of this lesion type corresponds to the percentage of connections to remove.

$$w_{ij} = 0 \qquad \textbf{Equation 4.1}$$

where $w_{ij}$ represents a connection between two arbitrary nodes in a feed forward network. Although, we will not be simulating the removal of a neuron, it is also possible to achieve this by simply ablating all connections leading to or from an individual neuron. An alternative, more direct method of removing a neuron is to set the internal activation of the ablated cell to zero (e.g. Medler, Dawson, & Kingstone, 2005). An analysis of lesion strategies involving ablation was performed by Ruppin & Reggia (1995) who investigated whether multiple focal lesions within an associative network provide more damage on tests simulating multi-infarct dementia than a single focal lesion of the same aggregate size. The concluded that in their model, multiple focal lesions show a larger deficit than a simple "sum" (single lesion of equivalent size) and reflects clinical studies suggesting that the main factors related to the prevalence of dementia after stroke to be infarct number and site (and not overall infarct size).

2) **Noise**

Neural systems are examples of natural systems where the elements are highly nonlinear, and subject to various noise levels (e.g. Netoff et al, 2004). Excessive noise however, produces a too-low signal to noise ratio leading to abnormal nervous system behaviour. Some authors appreciating the innate noisiness of neurons have applied this principle in their models. In simulating lexical access for example, Dell et al (1997) use an *intrinsic noise* variable replicating the innate noisy nature of the

nervous system. They also add a *noise* term, directly related to the activation of the net input, such that the higher the activation, the more noisy the signal. A similar technique is also used by Camperi & Wang (1998) who include a *white noise* term in their activation function. Simulating noise in a connectionist fashion can be achieved by adding a random value to the connection weight between two nodes.

In our simulations, we add a value between two bounds to every connection in order to control the effects in a rigorous and defined manner, and the severity of the lesion is determined by the relative difference between the upper and lower bounds.

$$w_{ij}(A) = w_{ij}(B) + Random(lower, upper) \qquad \textbf{Equation 4.2}$$

where *lower*, and *upper* define a range of random numbers from a uniform distribution, and *B*, and *A* represent *before*, and *after*, or pre and post lesioned values respectively. A uniform distribution ensures that all values in the desired range are equally likely. Braver, Barch & Cohen (1999) for example, simulated excessive disturbances in the dopamine system by increasing noise to a gating unit to five times that of the rest of the units. Our definition of lower and upper bounds requires some clarification. In many examples in the literature where noise is added to simulate disorder, an absolute value is simply added to the connection weights. Our testing approach uses multiple instances of the same network each representing different pseudo-patients. We believe that in order to provide an approach that allows similar noise *severities* to be applied across multiple networks, an awareness of the effect such noise may have on that network is required. For example, given two networks A, and B respectively, one could add random values between –0.01 and 0.01 to each connection. This may have a detrimental effect in network A, yet relatively no effect in network B. This may be due to the fact that in network B the error range is computationally insignificant relative to the average connection

weights and associated standard deviation. Our approach therefore, has been to multiply the noise severity by the standard deviation of weights in the network being lesioned. The larger the spread of weights, the more impact any given lesion severity should have on each connection weight thus normalising the effect of a given lesion severity when applied to more than one network. Thomas & Karmiloff-Smith (2002) used a similar method, adding noise from random distributions with specific standard deviations to their network's connections thus influencing the severity of damage. Samsonovich and Ascoli (2005) also added noise whose severity was defined by the magnitude of a standard deviation term. A refinement to Equation 4.2 is therefore:

$$Lower = \sigma_N V_L \qquad \textbf{Equation 4.3}$$

$$Upper = \sigma_N V_U \qquad \textbf{Equation 4.4}$$

Where $\sigma_N$ is the standard deviation of all weights in the network, and $V_L$ and $V_U$ are the relative lower and upper noise values respectively.

3) **Scaling of connection weights**

Our third lesion type scales down connection weights reducing each connection's contribution. Although we can find no disorder that provides a natural equivalent to scaling, we believe that it is worth investigation the validity of such an approach from a pragmatic stance. Bullinaria (2004) suggests that modelling massively parallel brain processes by simulating neural networks on serial computers is only rendered feasible by abstracting out the essential details and reducing the size of the networks. Where there are more hidden units and connections than actually required to perform mappings, graceful degradation can take place. For much smaller networks however, the effect of individual damage contributions can be large enough to produce wildly fluctuating performance on individual items. Therefore, in small

networks, ablating a single hidden unit or set of connections may disproportionately effect the overall mean contribution, not experienced in much larger networks. Bullinaria claims that global weight scaling does not suffer from such random fluctuations, and it can therefore be used to simulate a smoothed form of lesioning giving a reasonable approximation in small networks to what will happen in more realistic networks. As he points out, this procedure involves *approximating* the effects of focal damage by applying diffuse damage, and there are clear limits to the validity of the approximation. In our model, we apply this technique diffusely.

Equation 4.5 describes how a weight is scaled in order to change the contribution of that connection. This technique may not be appropriate for all network types due to the underlying nature of each. For example a Kohonen self-organising map would not be affected by this technique, since a competitive algorithm is used to determine the *winning* node. With all nodes equally scaled down, there can be no change in the winner.

$$w_{ij}(A) = sw_{ij}(B) \qquad 0.0 \leq s \leq 1.0 \qquad \textbf{Equation 4.5}$$

where $s$ is a scaling value. Lesion severity corresponds to the level of scaling applied to every connection with a value of s of 1.0 representing no scaling at all (i.e. retain 100% of the existing connection strength). One variation of this approach is described in Horn et al (1996). The authors perform what they term *synaptic compensation*, where residual synapses following *synaptic deletion* (i.e. ablation) due to Alzheimer's are scaled *up* by a common factor. To achieve this effect, we would use a combination of ablation, and scaling (on residual connections) with a value of $s$ in Equation 4.5 greater than 1.0. Evidence of synaptic compensation as a consequence of Alzheimer's can be found in more detail in Scheff & Price (1993).

4) **Constrained Potential**

This approach simulates sub optimal connectivity between synapses due to a particular type of axonal degradation, and can best be described as simulating the behaviour of an axon's ability to recover after damage to its myelin sheath. We believe this to be a novel lesion technique not found elsewhere in the literature.

Myelin is a sheath covering axons interrupted periodically by gaps, or *nodes of Ranvie*, exposing the axonal membrane. Excitation along the axon travels faster *across* these nodes bringing a concomitant increase in excitation velocity. An alternative to myelin would be to make nerve fibres larger. For example, in the squid, the largest axons are between 0.5 and 1.0 millimeter in diameter, and conduct at rates no faster than about 20 meters per second. In contrast, a myelinated axon 20 micrometers in diameter conducts at 120 meters per second (Nicholls et al, 1992:171). A further benefit of reducing axonal thickness is that it avoids packing problems and allows more neuronal structure in a confined space.

One common disease attributable to demyelinization is Multiple Sclerosis (MS), where the effects can appear in the central nervous system: encephalon and especially white matter, spinal cord and optic nerves. Demyelinization takes the form of cerebro-spinal fluid replacing myelin. There is a natural process of healing: a typical lesion expands to a maximum and then shrinks, thanks to remyelinization. Unfortunately this healing process is limited and becomes less restorative over time (e.g. Ray, Stoeckel, Malandain, Ayache, 2001). This suggests a loss of an ability to recover over repeated demyelinization episodes. An analogy may be applied to an elastic band, which over time loses its ability to return to its unstretched state.

Another disorder linked to myelin deficiency is hydrocephalus where patients have demonstrated a loss of myelin protein (e.g. Bradley, 2000, Van Hove,

Kishnani, Demaerel, Kahler, Miller, Jaeken, Rutledge, 2000) as well as myelin damage (Hähnel, Münkel, Jansen, Heiland, Reidel, Freund, Aschoff, Sartor, 1999). Hähnel et al describe the most likely mechanisms for axonal damage in hydrocephalus as being vascular changes causing chronic ischemia, kinking, and stretching of myelinated fibres.

From a connectionist view we might consider demyelinization as directly affecting the connection strength available between two connected nodes. For example, if a fully myelinated axon is assumed to deliver 100% of the connection strength, then progressive demyelinization might constrain this facility in a predictive fashion. The model described in Chapters 5 and 6 does not rely on precise timing and coordination of action potentials (temporal coding). It is conceivable however for multi-path functions (e.g. a dual-route spelling model), that one route slowing down due to focal damage would result in observable symptoms of damage.

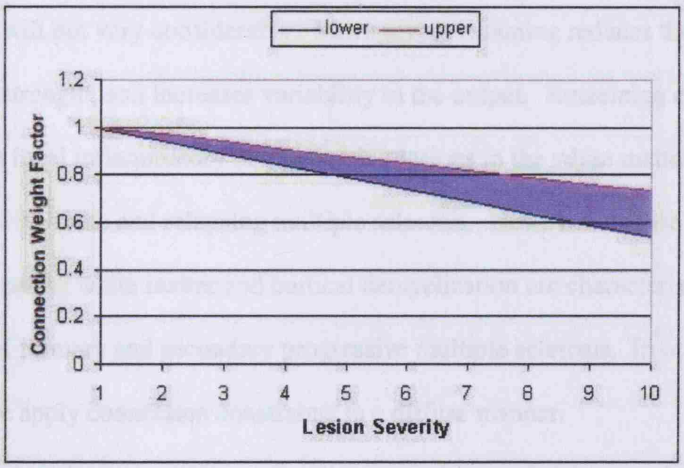



**Figure 4.2: Connection scales based on lesion severity. The shaded area highlights a sample minimum and maximum range possible at progressively increasing lesion severities.**

If connection strength is a function of the available artificial myelin, then one approach to simulating this is to define a minimum and maximum level of connection strength relative to the extent of damage. As described above, the processes of demyelinization and subsequent remyelinization have a longer-term

effect of not retaining previous connection strengths. Therefore, our approach to modelling this is to assume, that as lesion severity increases, the gap between the minimum and maximum strengths increases, and the maximum strength decreases, (See Figure 4.2).

$$w_{ij}(A) = w_{ij}(B) * Random(lower, upper) \qquad \textbf{Equation 4.6}$$

where *lower* and *upper* define the lower and upper bounds as a function of the severity of the disorder, and B, and A represent the weights before and after lesioning. The random function returns a number between these values taken from a uniform distribution. The overall connection strength is therefore calculated by *constraining* a perfectly functional connection relative to how stable the connection is deemed to be. At the lowest level of lesioning, there is a consistently strong connection. It is strong in the sense that we are taking almost full advantage of the required connection weights, and consistent in the sense that the weight for that connection will not vary considerably. More severe lesioning reduces the maximum connection strength, and increases variability in the output. Kutzelnigg et al, 2005 suggest that focal inflammatory demyelinating lesions in the white matter are found in patients with acute and relapsing multiple sclerosis. However, diffuse injury of normal-appearing white matter and cortical demyelization are characteristic hallmarks of primary and secondary progressive multiple sclerosis. In our model, we therefore apply connection constraints in a diffuse manner.

The previous lesion technique of scaling altered an artificial neuron's overall activation reducing it by a fixed amount, and was used to compensate for artefacts that may have been introduced in lesioning small networks. This technique differs in at least two areas. Firstly, it reflects aspects of known neuronal damage, namely

myelin damage. Secondly, it introduces an element of randomness while lesioning making if more physiologically plausible.

5) **Modification of the activation function**

In connectionist terms, an activation function simulates the depolarisation or hyperpolarisation of an artificial neuron, and reflects the sum activity of postsynaptic potentials feeding into the neuron. Excitatory postsynaptic potentials (EPSPs) are depolarisations and increase the probability of the postsynaptic neuron firing, and inhibitory postsynaptic potentials (IPSPs) are hyperpolarisations reducing the probability of a neuron firing. Connections to the artificial neuron thus reflect the excitation or inhibition arriving on neuronal dendrites. If the sum activity exceeds an internal threshold, then the neuron depolarises sending activity down its axon. This axonal activity is represented in artificial neurons by activation function values. Early models of artificial neurons used binary thresholds (e.g. McCullough & Pitts, 1943) simulating all-or-nothing depolarisations. Modern connectionists however generally use continuous differentiable threshold functions such as the sigmoid function (e.g. Rumelhart et al, 1986, Haykin, 1994). The outputs of these continuous functions do not reflect actual postsynaptic potentials in the brain. Biological postsynaptic potentials appear as roughly equal positive and negative potential fluctuations with amplitudes of around 0.1 mV (Dayan & Abbott, 2001). The output of a modern activation function however, is generally continuous and can be said to reflect the probability of a neuron firing. Connectionists may choose to use simple sigmoid functions, or may use more complex functions. Real neurons experience neuromodulation, which naturally modify neuronal synaptic potential, and a number of connectionist modellers have built this into their activation functions. In Camperi & Wang's (1998) model of visuospatial working memory, neuronal firing patterns

were produced where performance could be rendered robust against noise and distraction if neurons were endowed with cellular bistability. Their activation function included a gain term described as *synaptic drive*, which directly reflected a level of neuromodulation. Neuromodulators such as norepinephrine and dopamine for example, are often assumed to mediate the effects of attentional control of cognitive processing (e.g. Arnsten, 1998). Camperi and Wang showed that robustness against noise could be achieved by altering the activation function to ensure bistability. Conversely by modifying the gain term (and thus the bistability), the neuron can be shown to be prone to noise. Cohen & Servan-Schreiber (1992) modified a gain value with a constant bias as part of a simple sigmoid function simulating the effects of change of dopamine activity. With an increased gain, the neuron was more sensitive to excitatory input, and with a decreased gain, the neuron was more sensitive to inhibitory input. Usher & Develaar (2002) also simulated neuromodulation by altering a gain value within their activation functions directly affecting the contribution of synaptic inputs and leading to different output values. Later in the thesis, we examine the effect of using modified activation functions in our model. In brief, an activation function is modified by adding a *damping* term, constraining the activation function's full potential. We apply this technique in a focal manner when investigating the affect on the model's serial production mechanism. We will clarify this further in Chapter 6.

6) **Adding noise to the activation function.**

This technique is a minor modification to changing an activation function. It differs from the previous approach in that the activation function remains intact, and noise is added to it *after* the function has operated on all input. In our model this technique is applied in a focal manner.

In their model of dopamine control as a direct source of schizophrenic symptoms, Braver et al (1999) add noise to their model's activation state to simulate variability in processing. Burgess & Hitch (1992) added noise to the activation function within their competitive queuing model of verbal short-term memory, as did Houghton et al (1994), and Glasspool et al (2004, 2006) in their simulations of Graphemic Buffer Disorder. In our model, we add noise to an activation function between two bounds.

$$\alpha = F(I) + Random(lower, upper) \qquad \textbf{Equation 4.7}$$

Where $\alpha$ is the resultant activation, $F(I)$ is the logistic sigmoid function $F(x) = 1/(1+e^{-x})$, I represents all inputs to the node being processed, and *lower* and *upper* specify the range of noise to add to the activation function. Lesion severity corresponds to the difference between the lower and upper bounds with greater differences producing more noise.

One major difference between modifying an activation function and adding noise to an activation function is that the output values produced by adding noise can exceed the bounds imposed by the activation function limits. In our model we will apply this technique focally.

## 4.3 Lesion Methodology

Modelling the effects of disorder is usually demonstrated in the literature by applying individual lesion severities to simulate damage to the nervous system. For example, Glasspool et al (2006) lesioned their model of Graphemic Buffer Disorder by scaling connection weights by a factor of 0.58 to match the spelling performance of patient DA. Plaut (1995) also chose specific lesion locations and severities to demonstrate that double dissociations were achievable using a single connectionist module. His

investigations however, seemed to identify an *ideal* severity, providing results matching patient data, or desired results. Others have used a number of individual severities to simulating different patient types. In modelling acquired dyslexia, Hinton & Shallice (1991) used various noise and ablation severities to analyse how a range of lesions might affect their network. They argued that a benefit to testing multiple severities is that each combination may match specific patient behaviour as they claimed to do with patient AR. Our intent in applying multiple lesion severities to our model is to provide a structured approach to measuring the robustness of the model in the presence of damage. If for example, the model behaves consistently after applying a broad range of lesion severities, then it suggests that our results are not confined to a very narrow or prescriptive range of lesion severities.

With such a lesioning approach in mind, we have also ensured that in applying progressively larger severities of damage to our model, we do not inadvertently simulate relationships between lesion severities that one might expect say, with a progressive disorder. In other words, damage associated with each lesion severity should be independent, and unrelated. We might expect that pathological damage in the latter stages of a progressive disease for example, would also include damage that had occurred in the early stages of disease. In our model we avoid this ensuring that comparisons across lesion severities are comparisons of the *extent* of damage. Later in the thesis, we will also investigate the effect of lesioning specific areas within the model, to determine whether functionally important locations, have a corresponding effect when lesioned. Plaut & Shallice (1993) for example, describe how lesioning the direct pathway of their input network leads to an advantage in correct performance for concrete over abstract words, and Houghton & Zorzi (2003) lesion their lexical route in order to simulate characteristics of surface dysgraphia. We similarly intend to show that focal

lesioning can have a substantive effect. Table 4.2 summarises how each lesion type will be applied to our model, and how severity can be varied.

| Lesion Type | Parameters | Connections affected | Severity results from |
|---|---|---|---|
| Ablation | Percentage of connections to remove | Vary according to lesion severity | Number of connections removed |
| Noise | Lower and upper bounds of noise | All | The relative difference between both bounds |
| Scale | Scale value | All | Scaling down each connection by a value between 0.0 and 1.0. |
| Constrain | Lower and upper bounds | All | The relative difference between both bounds and the value of the upper bound. |
| Modify Activation Fn | Damping term | Those leading from the artificial neuron. | Extent of the damping value. |
| Add Noise to Activation Fn | Lower and upper bounds of noise | Those leading from the artificial neuron. | The relative difference between both bounds. |

**Table 4.2 : Summary of lesion types and severity strategies**

## 4.4 Conclusion

When connectionism was still novel, and cognitive simulations were generally constructed with symbolic models, modellers would usually treat their *black boxes* as many computer scientists would. In effect, they provided an input, modified parameters, and then analysed the outputs. The focus seemed to be more on clarifying whether computers could simulate visible human behaviour than in the nature of the internal mechanisms responsible for that behaviour. Today, more information exists as to how the nervous system actually works. In conjunction with more computing power, modellers have more opportunity to simulate the internal mechanisms of these black boxes and determine whether a connectionist approach not only reflects intact behaviour, but whether applying damage in ways found in real nervous systems provides comparable effects when applied to artificial systems.

We have summarised a number of lesion techniques, showing similar affects in artificial models to those found in biological networks in the presence of damage. Some of these lesion types are commonly used such as ablation and noise. However, we introduced at least one technique which we believe is novel, namely constraining connectivity in a manner similar to myelin breakdown. Despite the broad adoption of such techniques in the literature, their limited biological realism suggests a cautious approach to any conclusions one might make regarding the nature of the nervous system. Their use is primarily an exercise in mathematical modelling, and the modeller should consider whether these techniques are justifiable in the context of the disorder being researched.

Our next chapter investigates and develops further, a connectionist model that has been shown to simulate aspects of both Graphemic Buffer Disorder and Deep Dysgraphia using some of the lesion types described her

# 5 Towards a Model of Spelling

## 5.1 Introduction

In Chapters 2 and 3, we described models of spelling and how these may provide an account for symptoms of graphemic buffer disorder (GBD) and deep dysgraphia (DD). Subsequently in Chapter 4 we introduced a number of artificial lesion techniques that crudely reflect damage found in the nervous system. In this chapter, we examine an architecture composed of separate yet related sub-networks that has been shown (Glasspool, Shallice & Cipolotti, 2006) to simulate aspects of spelling dysfunction, namely GBD and DD, and to a lesser extent unilateral neglect. We primarily build on the model described in Glasspool et al and due to our numerous future references we will be summarise their work as the "GSC" model. The GSC model is itself based on Competitive Queuing (CQ) principles described in the literature (e.g. Houghton, 1990, Houghton, 1994, Houghton, Glasspool & Shallice, 1994, Shallice, Glasspool & Houghton, 1995, Glasspool, 1998, Ward & Romani, 1998, Glasspool & Houghton, 2005). In their most basic form, CQ models propose that serially ordered memories are comprised of learned chunks, each consisting of a limited number of behavioural units, linked by learned connections to units at a higher level. At recall, a number of nodes are activated in parallel by excitatory input from a sequence level with a gradient of activation over them such that the sooner an item is to be generated, the more active it is. Items compete for control of output, and the winner is inhibited preventing its participation in competition for the next response item (Houghton, 1994). It fundamentally differs from a number of other approaches such as chaining (Elman, 1990) by providing an overall control mechanism. A key criterion in determining whether a model reflects human behaviour is not only whether that model can be

considered to have simulated working behaviour, but whether it also provides comparable behaviour to human subjects when damaged. Chaining for example, which uses currently active items as key determinants of future items, may be problematic since a letter's omission may prevent it reappearing elsewhere in a predictive fashion such as with transpositions (see however Botvinick & Plaut, 2004). In addition, an omission may prevent the following correct letters from appearing due to the omission not *priming* them. An overall control mechanism however, allows spurious errors to have minimal effect on the overall serial order of the word or sequence being produced.

In this chapter we will revalidate certain operational aspects of the GSC model so as to expand its functionality and to refine or resolve issues we believe problematic in the original design. This chapter does not present results as these are provided in substantial detail in Chapters 6 and 7.

## 5.2 Competitive Queuing Principles

Glasspool & Houghton (2005) describe three features typically present in CQ models:

1) *A set of refractory representations*: These representations are distinct and competitive in that for the most part, only one can win for a time frame in question. Secondly, the responses are generally refractory such that that sequence item becomes temporarily unavailable for further use.
2) *Parallel response activation and activation gradient*: Responses in a target sequence are activated in parallel at the beginning of recall. However, viewed over time within the sequence, one would see a gradient of activation such that the sooner a response is to be produced, the more active it is. In effect, it is as though the relevant response prepares itself for production.

3) *A competitive output process*: The currently most active response is deemed the *winner* on exceeding a threshold, and becomes self-refractory

As our model builds on the original GSC model, we adhere to these original principles and will describe how our changes compare to CQ models described by authors such as Burgess & Hitch (1992), Houghton, Glasspool & Shallice (1994), Shallice, Glasspool & Houghton (1995), Ward, Olson & Romani (1998), and Glasspool & Houghton (2005). Areas where we have concentrated our research include:

a) Providing a broad range of error metrics to allow comparisons with other results in the literature (Caramazza, Miceli, Villa, Romani, 1987, Caramazza & Miceli, 1990, and Cipolotti et al, 2004).

b) Catering for consonant/vowel identification (e.g. Glasspool 1998, Houghton et al, 1994, Glasspool & Houghton, 2004) as well as geminate identity (Houghton et al 1994, Glasspool & Houghton, 2005).

c) Examining consonant/vowel complexity and associated error behaviour. Orthographic complexity and its influence on the error behaviour have been discussed in the literature (e.g. Caramazza & Miceli, 1990, Jonsdottir, Shallice & Wise, 1996, Kay & Hanley, 1994, Tainturier & Caramazza, 1996). We provide a means of comparing the influence of orthographic structure on the model to patient error behaviour.

d) Exposing the model to a broader range of lesion types and severities, our objective being to determine how robust the model is in the presence of damage.

e) Provide the basis for an error analysis workbench extending the scope of comparisons and hypothesis testing.

f) Re-examining some of the underlying model's assumptions and making modifications where we believe they can be substantiated.

One objective of the GSC model was to test the assumption that although GBD and DD may be different disorders, they share a predictable set of symptoms that can be attributed to properties associated with the graphemic output buffer. They were termed GBD type-A and type B respectively. This was achieved by joining two sub-networks, each trained in such a way as to produce type A and type B behaviour when selectively lesioned. We expand on the analyses applied to the original model in order to gauge whether two networks are indeed required to simulate all aspects of these distinct disorder types. We discuss the foundation of this functionality and the relevant metrics in this chapter and then expand on these more fully in Chapter 6. A concise description and foundations for the model are available in Glasspool (1998), Glasspool et al (2006), Glasspool & Houghton (2005), and Shallice et al (1995). We will therefore only concentrate on describing in detail those areas of our model that we feel are sufficiently different from the original, warrant discussion as being fundamental to the model's architecture, or require clarification. In addition, we have not expanded on the new model's training performance compared to the original; the focus of this chapter is in establishing a framework for analysing behaviour in the presence of damage, not training.

## 5.3 Functional attributes of a working model

If the claims by Cipolotti et al (2006) are correct, then we would expect a working model of spelling producing type A and B GBD to have a number of characteristics.

*General Attributes*

1. The model must be able to spell words correctly by processing one letter at a time.
2. It should exhibit a word length effect, such that longer words are more susceptible to errors than shorter words.

3. Lesioning selective parts of the model should provide *typical* errors for type A and type B patients, based on the locus of lesioning.
4. The model should allow geminate identification and produce typical doubling errors.
5. The model must show a consonant/vowel (CV) effect, which would manifest itself as CV preservation in transpositions and substitutions.

*Type-A attributes – Lesioning applied in Graphemic Output Buffer area*

6. Errors should demonstrate a bow-shaped error curve, errors being more prevalent in the middle of words than at either end.
7. There should be no significant difference between abstract and concrete error rates.
8. There should be no significant difference between low and high frequency errors.

*Type-B Attributes – Lesion applied in Semantic area*

9. Errors should show a serial effect, being more prevalent towards the end of words.
10. There should be significant differences between abstract and concrete errors.
11. There should be significant differences between low and high frequency errors.

*Architectural Attributes*

12. The model should allow a broad range of lesion severities, loci of lesioning, and lesion types to be applied, allowing a detailed investigation of network behaviour.

## 5.4 System Architecture

The GSC model assumes that both type-A and type-B GBD arise from separate yet related faculties and predicts that lesioning different areas of the network in a specific manner should provide behaviour corresponding to that shown by both types of patients. Where a model can be viewed as containing functionally dissociable components, then

lesioning selective parts of the model is a strategy one may consider to evaluate the effect of damage. Hinton & Shallice (1991) for example, used a model containing semantic and graphemic representations to produce symptoms of deep dyslexia by selective lesion of different parts of the network, and Wright & Ahmad (1995) used selective lesioning to simulate aspects of aphasic naming. Before exploring potential areas for improvement or change in the model, we will first describe relevant aspects of the original GSC model.

### 5.4.1 A Brief Overview of the GSC model

The original GSC model (Figure 5.1) contains two networks, each held accountable for the source of either type-A or B GBD behaviour. At a high level, Glasspool et al viewed the GOB as a CQ system for generating sequences of letter identities when receiving input representing the identity of a word to be spelled, and the semantic activating system as responsible for providing that word identity.

CQ models use a control mechanism or *context* to manage the serial order of constituent items and the GSC model uses the context introduced by Burgess & Hitch (1992). This context is shown in the box titled 'Serial position representation' in the top left of Figure 5.1. The context and word identity are fed forward through a hidden layer where every letter output node (A-Z) produces an activation. The letter with the highest activation is deemed to *win* and is output as the *correct* letter. In addition, a consonant/vowel (CV) status is produced, which after training reflects the CV type of the correct letter.

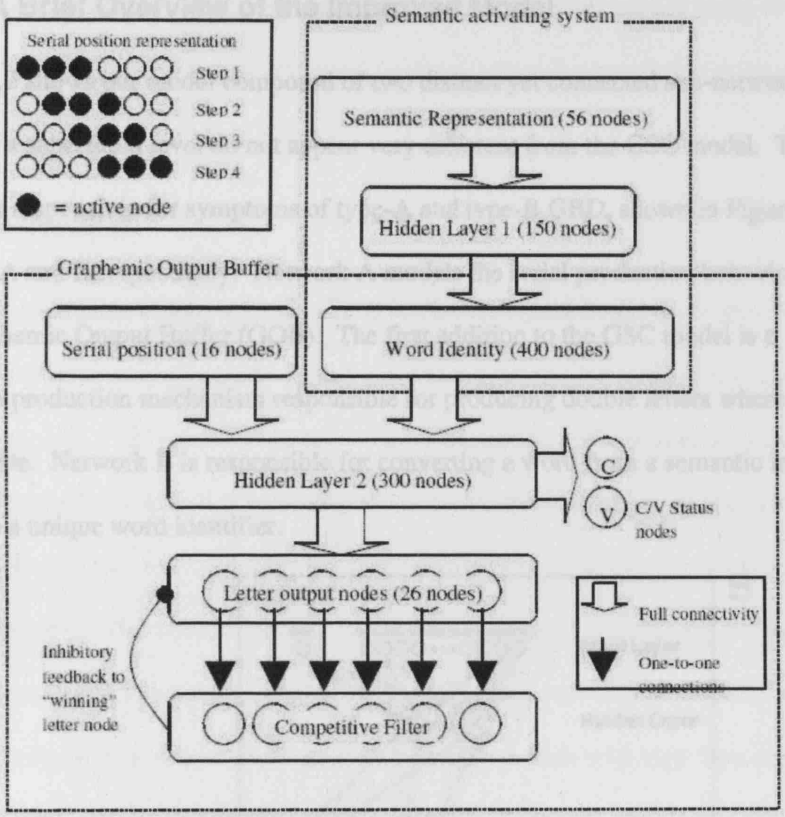



**Figure 5.1: The original GSC model by Glasspool et al (2006).**

As we examine later in the thesis, the CV status also appears to have a distinct effect on the types of erroneous letters produced during transpositions and substitutions. After a letter has been produced, it is temporarily inhibited preventing it from being produced in the following serial position. Note that the original model lacks any ability to deal with double letters (or geminates). We provide a resolution to the problem of dealing with geminates using an approach similar to recent work by Glasspool & Houghton (2005), and expand on this in considerable detail in Chapter 7. Unlike the Graphemic Output Buffer network, damage to the Semantic Activating System should produce significant error differences between abstract and concrete, and low and high frequency words.

### 5.4.2 A Brief Overview of the Improved Model

Figure 5.2 shows our model composed of two distinct yet connected sub-networks, which at a superficial level do not appear very different from the GSC model. The networks responsible for symptoms of type-A and type-B GBD, shown in Figure 5.2 are labelled A and B respectively. Network A models the serial production behaviour of the Graphemic Output Buffer (GOB). The first addition to the GSC model is a geminate production mechanism responsible for producing double letters where appropriate. Network B is responsible for converting a word from a semantic input vector to a unique word identifier.

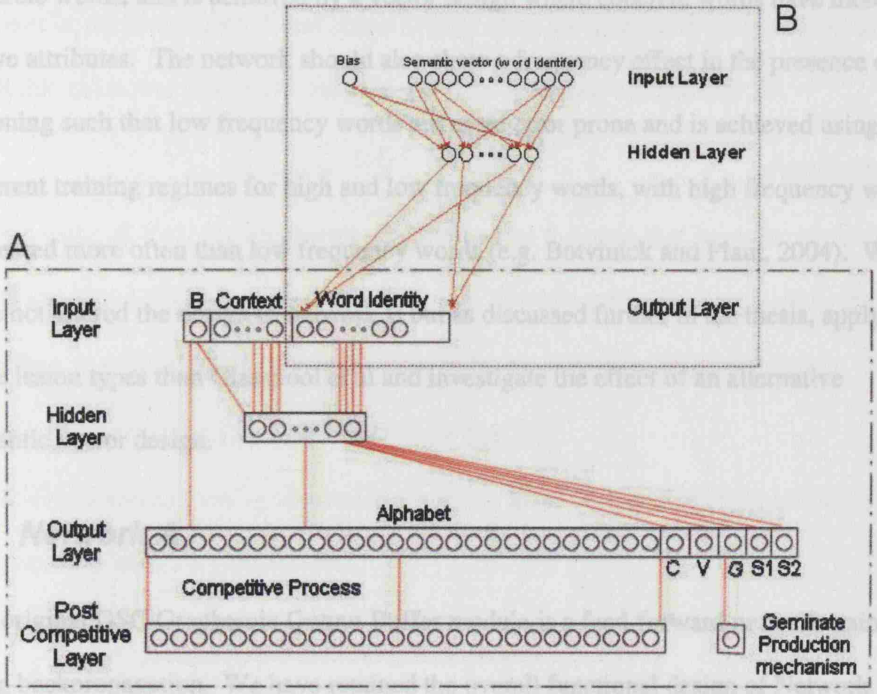



**Figure 5.2: The network showing two joined sub-networks. Network A is primarily responsible for Graphemic Output Buffer simulation, and Network B for Deep Dysgraphia simulation.**

A vector representing semantic information is presented at the input layer of network B and cascaded down via a feed forward mechanism where the resultant word identity is included in the input for Network A. The word identity is combined with a *context* value, which is used to order a word's constituent characters, and fed forward through

Network A resulting in the serial production of characters for the word being recalled. We will first describe each network briefly before examining constituent components in more detail.

## 5.5 Network B

As can be seen from Figure 5.2, network B has a very simple multi-layer structure with a single hidden layer and is trained using back propagation. It translates a semantic input vector into a unique word identity. The composition of the semantic vector ensures that in the presence of lesioning, there should be more errors in abstract than concrete words, and is achieved by a vector design where concrete words have more active attributes. The network should also show a frequency effect in the presence of lesioning such that low frequency words are more error prone and is achieved using different training regimes for high and low frequency words, with high frequency words presented more often than low frequency words (e.g. Botvinick and Plaut, 2004). We have not altered the design of network B but as discussed further in the thesis, apply more lesion types than Glasspool et al and investigate the effect of an alternative semantic vector design.

## 5.6 Network A

The original GSC Graphemic Output Buffer module is a feed-forward network trained using backpropagation. We have retained the overall functional design of Network A, yet there are some areas where improvement has been investigated, which we examine in more detail here. We will start at the input layer progressing to the post-competitive layer highlighting differences in each model where applicable. However, before investigating the nature of letter production, it is worth considering what mechanisms can be used to cease production.

### 5.6.1 When to Stop Letter Recall

Houghton et al (1994) describe three alternative methods for cessation of spelling:

1. *Stopping when the correct number of graphemes has been recalled.*

   One implication of this method is that it does not allow recall of words with a length longer than the original, such as additions early in the word. In such cases, recall will cease before the final letters have a chance to be produced.

2. *Stopping when the overall activation level of all letter nodes falls below some threshold.*

   Ward et al (1998) used this approach to help accentuate the presence of fragments (two or more omissions), and chose to stop when the combined activation of all letter nodes was below a certain threshold as described in Equation 5.1.

   $$\sum_{n=1 \text{ to } 26} |A_n| < 0.42 \qquad \textbf{Equation 5.1}$$

   His value of 0.42 was empirically determined on the basis that it produced the most efficient stopping behaviour for his corpus. Too low a threshold may cause trailing letters (e.g. WOMAN → WOMANW), and a too high threshold may cause premature stopping (e.g. BACHELOR → BACH).

3. *Adding a special 'end of sequence' grapheme to all words, ceasing recall when this grapheme wins the competition.*

   This approach, used by Glasspool (1998), Botvinick & Plaut (2004), and in the original GSC model, is also used by us in the current model. Glasspool (1998) used the term *stop-state* to denote this as an end-of-sequence grapheme. This seems appropriate since there is no real grapheme identity, and a winning value simply indicates end of recall. Stop states are not shown in Figure 5.1, yet the GSC model does indeed use two trailing positions and is shown in the new model in Figure 5.2 labelled S1 and S2. The more stop states, the more extra characters can be added to

erroneous words. It is possible to use any number of stop states to allow for several additions to the word in question, and the stop-state is learned like any other letter. Therefore a four letter word like CANE with two stop states, would be encoded as 6 letters 'CANE!!' where '!' is interpreted as a stop state. If the stop state is not close enough to its desired value, the most active letter is produced, thus creating trailing additions.

A benefit of using stop states is that error in recall allows additions after the natural end of a word. In our tests, we observed perseverative errors such as WOMAN → WOMANAN. A benefit of the Ward approach over stop states is however that stop-states constrain the maximum number of extra letters to the number of stop states presented during training. The Ward approach allows any number of extra characters as long as the sum activation of all letters remains above a given value. A deficiency of the Ward model however, is that empirical research is required to find an appropriate stopping level and this will be network and corpus sensitive. We now investigate the mechanisms underlying letter production.

### 5.6.2 The Input layer

In addition to the bias node labelled **B**, Network A's input comprises two other node groups (see Figure 5.2). The first holds a unique reference to the word being examined, and is called the *word identity*. The second contains what Houghton (1994) terms a *time varying* or *control signal*. The term *context* has been used by Burgess & Hitch (1992) and Ward (1997) and is our preferred term. We now discuss each in detail:

#### 5.6.2.1 Word Identity

The word identity comprises a number of nodes uniquely identifying each word being learned or recalled. The composition of the word identity nodes, as with the GSC

model requires each feature vector (i.e. corpus member) to have four (from a total of 400) randomly assigned active nodes. The fact that each vector has an equal number of active nodes has two implications. Firstly, all corpus members are expected to be equally susceptible to damage in terms of the effect of lesioning word-identity level. Therefore, lesioning should not produce any concreteness related errors. Secondly the chance of feature vectors overlapping in a systematic fashion is small, so there is no reason to expect plausible semantic errors across word identities.

### 5.6.2.2 Letter Context

These nodes describe the relationship between successive letters being spelled, and thus affect both the serial position of a word's constituent letters and their propensity for error. The literature shows at least two methods of achieving this: *Initiator/End* (IE) by Houghton (1990), and *Moving Window* (MW) by Burgess & Hitch (1992). We apply a third method, which we call *simple positional*, which unlike the first two methods should produce no serial position curve typical in GBD patients. Simple examples of these methods, or *contexts*, as they have also been termed are shown in Table 5.1 and we now describe each in more detail.

| Word | Letter | *p* | Context Type | | | |
|---|---|---|---|---|---|---|
| | | | *Positional* | *IE* | | *Moving Window* |
| CANE | C | 1 | **1** 0 0 0 0 0 | 1.000 | 0.216 | **1 1 1 1** 0 0 0 0 0 |
| CANE | A | 2 | 0 **1** 0 0 0 0 | 0.600 | 0.316 | 0 **1 1 1 1** 0 0 0 0 |
| CANE | N | 3 | 0 0 **1** 0 0 0 | 0.360 | 0.600 | 0 0 **1 1 1 1** 0 0 0 |
| CANE | E | 4 | 0 0 0 **1** 0 0 | 0.216 | 1.000 | 0 0 0 **1 1 1 1** 0 0 |
| CANE | ! | 5 | 0 0 0 0 **1** 0 | 0.129 | 1.000 | 0 0 0 0 **1 1 1 1** 0 |
| CANE | ! | 6 | 0 0 0 0 0 **1** | 0.077 | 1.000 | 0 0 0 0 0 **1 1 1 1** |
| TOP | T | 1 | **1** 0 0 0 0 0 | 1.000 | 0.360 | **1 1 1 1** 0 0 0 0 0 |
| TOP | O | 2 | 0 **1** 0 0 0 0 | 0.600 | 0.600 | 0 **1 1 1 1** 0 0 0 0 |
| TOP | P | 3 | 0 0 **1** 0 0 0 | 0.360 | 1.000 | 0 0 **1 1 1 1** 0 0 0 |
| TOP | ! | 4 | 0 0 0 **1** 0 0 | 0.216 | 1.000 | 0 0 0 **1 1 1 1** 0 0 |
| TOP | ! | 5 | 0 0 0 0 **1** 0 | 0.129 | 1.000 | 0 0 0 0 **1 1 1 1** 0 |

**Table 5.1: Values for each context type for the words CANE and TOP. A! represents a stop state.**

#### *5.6.2.2.1 Simple Positional Context*

This context is represented by a single active state indicating the desired location of a letter at a point in time with all other letter locations remaining inactive. Although words will be spelled correctly, this context does not predict a serial position effect often seen in patients with GBD. A key feature of this context type is that there is no inherent propensity for error for letters in medial serial positions.

#### 5.6.2.2.2 *Moving Window* Context

Introduced by Burgess & Hitch (1992), this context type assumes that neighbouring letters are related due to their proximity. Thus, close letters should be more likely to transpose than letters much further away from each other. This is achieved by ensuring that neighbouring letters have similar (overlapping) features. All nodes in the context section up to but not including the current letter are inactive, and then a number of nodes equal to a *window* size are set active. The window is a group of nodes that are partly shared with adjacent letters and the larger the window, the more opportunity for overlap. Following the window, all nodes to the end of the context section are set inactive. In effect, a *moving window* of activation is created reflecting the expected position of the current letter. In the presence of lesioning, neighbouring positions which overlap will have similar context values and thus be more prone to transposition. Letters at the beginning and end of words share fewer overlapping context positions than those towards the middle of words and should be less prone to transpositions. Note that the positional context described earlier is a special example of the moving window context having a window size of one, and thus no opportunity for neighbouring features to overlap.

#### 5.6.2.2.3 IE *Context*

This technique introduced by Houghton (1990) has been used to simulate a variety of order-related symptoms (e.g. Houghton et al, 1994, Glasspool & Houghton, 2005) and is composed of an 'I' (initiator) node and an 'E' (end) node. The 'I' node has a high value at the beginning of words and degrades exponentially as the letter position increases. The E node has a low value at the beginning of the word and increases exponentially as the letter position increases (see Figure 5.3). The steeper the gradient between positions, the less influence lesioning should have. Therefore more errors would be expected in medial positions where the difference between adjacent letters is not as high as at the extremes.

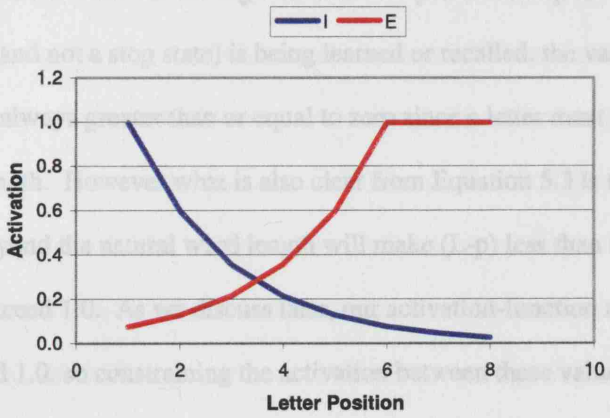



**Figure 5.3: I and E values for a six letter word with two stop states.**

We use the formula from Glasspool (1998) describing the I and E curves as follows:

$$I = F^{p} \qquad \textbf{Equation 5.2}$$

$$E = F^{L-p} \qquad \textbf{Equation 5.3}$$

Where $F$ is a decay parameter (of value 0.60) defining the steepness of the exponential curve, $p$ is the serial position of the current letter, and $L$ is the length of the word being

learned or recalled. Using a known word length to calculate serial position may be subject to critique yet Houghton (1994), suggests that as the word to be learned might be thought of as being scanned from left to right with the E-node activation increasing as an exponential function of the *perceived* distance from the end, then this may not be so implausible. Moreover he describes how the length term can be removed completely as long as the network is able to remember how active the E node should be at the start of recall. The I node is a function of natural decay, and the E node is the *reverse*, which can be derived directly from the E node. The IE values in Table 5.1 show a clear mirror image based on serial position (p).

Figure 5.3 also shows that the E value is constrained to a maximum value of 1.0. We do this in order to cater for the high activation expected at stop-state positions. Where a letter (and not a stop state) is being learned or recalled, the value of (L-p) from Equation 5.3 is always greater than or equal to zero since a letter must exist within the natural word length. However what is also clear from Equation 5.3 is that referencing any position beyond the natural word length will make (L-p) less than zero, causing the value of E to exceed 1.0. As we discuss later, our activation-function range lies between 0.0 and 1.0, so constraining the activation between these values is in keeping with the model's natural limits.

### 5.6.3 Controlled Recovery and Inhibition

Rumelhart and Norman (1982) arrived at two conclusions related to geminates. Firstly, there seems to be a special schemata signalling the existence of doubles. Secondly, the need for such a special schema implies a difficulty in having the regular schema produce the double. We investigate this phenomenon in substantial detail in Chapter 7, but it seems logical to refer to it as an introduction of a natural inhibitory mechanism, which would prevent a letter appearing immediately following its production.

To clarify the effect that localised inhibition may have on spelling, consider what would happen where inhibition is too strong, or too weak. Vousden & Brown (1997) describe a failure to recall a repeated item correctly as a *repetition omission* (e.g. ABCBD → ABCD), and production of a repeated action in error as a *repetition insertion* (e.g. ABCD → ABCB). This form of repetition should not be confused with geminate production; the repetition does not have an immediate doubling effect but occurs later in the word. In the extreme case where inhibition is complete and permanent, it would be impossible for a letter to appear twice in the same word, thus leading to many repetition omissions, and no repetition inserts. Conversely, in the absence of any inhibition, we would expect many repetition insertions and possibly many false geminates.

Houghton et al (1994) use an inhibitory mechanism reflecting the temporary suppression of previous neuronal activity. For example, for a few milliseconds just after a cell fires, it may be virtually impossible to fire again and is known as the absolute refractory period. For a longer interval known as the relative refractory period lasting up to tens of milliseconds after a spike, it is more difficult for a cell to fire (Dayan & Abbot, 2001). Thus the more recently a letter has appeared the more difficult it should be produce it again. One problem with such an approach is that geminates are impossible to produce without resorting to a specific doubling mechanism, which we introduce in the new model. Further, the inhibitory mechanism should be of an adequate level so as to avoid excessive repetition additions and omissions. We achieve this by scaling down a letter's activation relative to how recently it appeared as shown in Equation 5.4.

$$s = 1 - f^{p} \qquad \textbf{Equation 5.4}$$

where $f$ is a scaling factor, $p$ is the number of serial positions since the last occurrence of that letter, and $s$ is our activation scale. The new activation $a'$ is therefore calculated by multiplying the original activation by the scale.

$$a' = sa \qquad \textbf{Equation 5.5}$$

Figure 5.4 reflects a number of possible scale curves (Equation 5.4) based on the number of elapsed characters since a specific letter was produced. For example, when recalling the word 'TRAIN', in position four, the letter 'I' will have just been presented. The number of elapsed characters for 'I' in position five is therefore 0. It is possible that an 'I' will have the highest activation on recall at position five, but this will be multiplied by 0.0, ensuring that it cannot win. After production of the letter 'I', the elapsed characters for letters 'A', 'R', and 'T' are one, two, and three respectively. Figure 5.4 shows four curves based on different scaling factors ($f$), where larger factors make it more difficult for a letter to reappear close to its last presentation position.

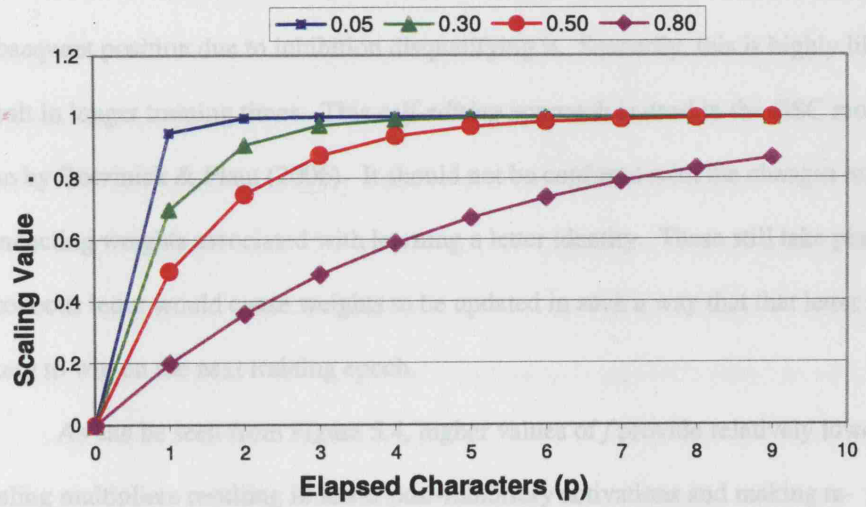



**Figure 5.4: Graphical representation of the different effects of various scaling factors**

Every scaling factor will inhibit the immediate recall of a letter (i.e. a double), but larger factors will inhibit recall of a letter for longer periods. Figure 5.4 for example, shows

that for a letter that was last produced two positions prior to the current position, the maximum activation possible with a scaling factor of 0.80 is 0.36. Unless letters with such low activations were to be eligible for production, it would become impossible to learn any corpus with words containing repeating letters any closer than two letters apart such as CEDER and BANANA. Conversely, a scaling factor of 0.05, ensures that a letter cannot be doubled, yet permits its reappearance relatively soon assuming that the letter's activation is sufficiently high prior to scaling. One effect of inhibition is that it may prevent production of the correct letter. The letter that should have been produced in the current position may have been erroneously produced in a prior (possibly the most recent) position. During training, a supervised correction procedure is used so that the letter that *should* have been produced is more likely to be produced next time. Inhibition is applied to the letter that *should* have won rather than the letter that did win. The implications of not using such a technique are twofold. Firstly, an erroneous letter produced earlier in the word may result in difficulty producing the correct letter in a subsequent position due to inhibition disqualifying it. Secondly, this is highly likely to result in longer training times. This *self-editing* approach is used in the GSC model and also by Botvinick & Plaut (2006). It should not be confused with the changes made to connecting weights associated with learning a letter identity. These still take place. An erroneous letter would cause weights to be updated in such a way that that letter is less likely to win on the next training epoch.

As can be seen from Figure 5.4, higher values of $f$ provide relatively lower scaling multipliers resulting in lower post-inhibitory activations and making re-presentation more difficult. A language such as English often repeats a letter as soon as two characters away. We are therefore constrained to the range of $f$ values we can use, since an overly restrictive value may make plausible recall impossible. The activation

of the most active letter is multiplied by this scaling factor to ensure non-reoccurrence followed by progressively more opportunity to reoccur relative to its subsequent recency. Following output of a letter, the corresponding letter identity node becomes refractory and is briefly unavailable for further output. In CQ experiments described by Houghton et al (1994) and the GSC model, an inhibitory technique called *kickback* is used. Kickback reduces the activation of the most recently presented letter to a negative value allowing it to recover gradually. It prevents the reappearance of the most recently produced item since the previously assigned negative value reduces the chances of that letter winning in the following position. The activation function range for Houghton et al's model was between -1.0 and 1.0.

$$A_i(t) = \begin{cases} net_i & \text{if } A_i(t-1) \geq 0 \\ net_i + rA_i(t-1) & \text{otherwise} \end{cases} \qquad \textbf{Equation 5.6}$$

where $r$ is a parameter governing the rate of recovery from inhibition, $A_i$ represents the activation of the letter node and $t$ represents a relative time-step (or serial position). As with the GSC model, we use a sigmoid function with a range between 0.0 and 1.0 so kickback to a negative value seems out of place and somewhat inelegant. A working GSC model shows that backpropagation is able to deal with kicked back activations. However, excessive effort by the backpropagation algorithm is almost certainly required for a letter to reappear only two positions later. Further, in the absence of stimulation, Houghton et al's approach allows a node's activation to gradually decay to zero (i.e. midway between -1.0 and 1.0). With the GSC model a node is free to maintain any activation as long as spelling performance is perfect during training and recall. This is discussed in more detail (section 5.11.1) when we describe the lazy update algorithm (Glasspool, 1998).

## 5.7 Letter Production

The GSC model separates the functions of *activating* a word's constituent graphemes (Output Layer), and a process by which these graphemes are *selected* (Competitive Layer). The approach used in the model of separating both these functions is supported by data from two patients, JRE and RSB (Rapp & Kong, 2002). Patient JRE exhibits a dissociation between difficulties in spelling words and high accuracy in a "single letter probe" task. This task requires her to indicate whether a particular letter exists in the target word. Conversely, patient RSB shows difficult with spelling and the single letter probe task. The authors argue that both spelling and single letter probe require adequate and sustained activation of orthographic representations, whereas spelling additionally requires the serially ordered selection of individual letters. They therefore conclude that RSB's deficit originates in the activation component , whereas JRE may suffer from damage to the selection component.

## 5.8 CV Status

Several studies of graphemic buffer deficits have shown that information about specific letter identities can be lost while knowledge of the letter's CV status is preserved suggesting that letter identity and CV status may be represented separately. A number of cases (Miceli, Silveri, & Caramazza, 1985, Caramazza & Miceli, 1990; Cubelli, 1991, McCloskey et al, 1994, Miceli et al 1995, Miceli, Capasso, Benvegnù & Caramazza, 2004) showed substitution errors often occurring within class, in other words, consonants being substituted for consonants and vowels being substituted for vowels. For example, errors like DECINE (tens) → CECINE were common, but errors like SCUOLA (school) → SCLOLA were very rare (Caramazza & Miceli, 1990). Patient LiB (Cotelli, Abutalebi, Zorzi and Cappa, 2003) however showed a particularly

strong case of within class substitutions. Of all errors, 97% represented substitutions, and all of these respected consonant/vowel status, and 85% of the errors affected vowels. In substitutions made by CW (Cubelli, 1991), 99% involved vowels for vowels and consonants for consonants.

If CV status and grapheme identity are represented independently, then information on letter identity may be lost, but knowledge of whether that letter is a consonant or a vowel may be retained. Thus, CV status may be sufficient to ensure that the incorrect letter preserves the CV status of the target. Like the GSC model, we incorporate a separate representation for consonant and vowel statuses. Network A in Figure 5.2 has two output nodes used for consonant and vowel identification. One indicates a letter being a consonant, and the other indicates a letter being a vowel. Each has an active status in the presence of the other being inactive. Glasspool and Houghton (2005) use a slightly different approach where CV status is used as input to the letter being learned. In chapter 6, we demonstrate that despite CV status being an *output* value associated with the currently active letter, the model does indeed show that consonants and vowels substitute and transpose with letters of the same type.

## 5.9 Letter Thresholds

When the system feeds activity forward, the *winning* letter is that with the highest activation at the Output Layer. This is normally the correct letter, but in the presence of lesioning could be any letter from A to Z. Being the most active letter is only the first stage in letter production as the letter's activation must also be sufficiently high to merit being shown. Figure 5.5 shows the typical activation function produced by all output neurons and three *thresholds*.

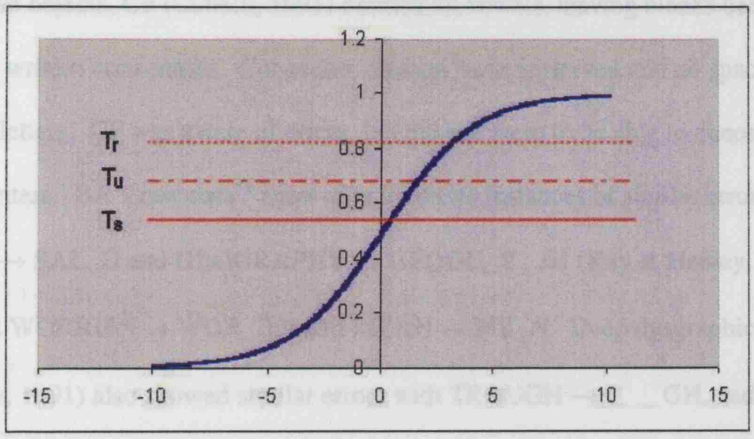



**Figure 5.5: The model's activation function when compared to various sample thresholds. See text below, for a discussion of the functions of $T_r$, $T_u$, and $T_s$.**

The GSC model considers a threshold, as a value that an activation function must reach for spelling production to operate successfully. There are three relative conditions against which letter activation can be compared:

1. *Above or equal to the letter Threshold $T_r$*

   The most active letter is produced even if it is incorrect. During training, if the activation of the desired letter is not at least equal to $T_r$, then that letter is reinforced to increase the probability of it appearing correctly in that position.

2. *Below the letter Threshold $T_r$ but above or equal to the stop threshold $T_s$*

   No character is output and recall continues. This is equivalent to a letter omission.

3. *Below the stop threshold $T_s$*

   Recall of the current word ceases.

We also investigated an additional threshold, which we term the *unknown* threshold $T_u$. Patients have been reported (e.g. Nolan & Caramazza, 1983, Miceli, Silveri & Caramazza, 1985, Katz 1991, Cubelli, 1991) to leave blank spaces between correctly written letters. For example, when asked to write his name and the name of his town

and of five objects, CF (Cubelli, 1991) omitted all vowels, leaving blanks between correctly written consonants. Consonant clusters were preserved and no space was left between letters. CF was aware of errors, but did not seem to be able to choose the correct letters. BA's raw data[18] showed at least two instances of similar errors including SALAD → SAL_D and GEOGRAPHY → GEOGU_Y. JH (Kay & Hanley, 1994) produced WORRIES → WOR_IES and MEAN → ME_N. Deep dysgraphic patient HR (Katz, 1991) also showed similar errors with TROUGH → T___GH, and GONE → GO_E. Nolan and Caramazza (1983) simply classified FV's unknowns (e.g. LENGTH → LENG_H, NURSE → NU_SE) as deletes, but there seems to be merit in measuring unknowns as distinct from deletes as this distinguishes between patients who are unable to choose the target letter (e.g. TASTE → T_ST_) and those who would make incorrect choices leading to letter substitutions (e.g. TASTE → TOSTA). Figure 5.5 shows, the unknown threshold $T_u$ lying between $T_r$ and $T_s$. An activation between $T_u$ and $T_r$ suggests that a letter merits presentation, but is not active enough to be identified specifically, thus producing a blank space or a '?' character. In preliminary tests of the model, we were able to produce errors such as those shown by patients BA, JH, HR, and FV. We believe it beyond the scope of the thesis to investigate this in substantial detail yet it is worth considering for future work.

Our model also allows different thresholds for each letter. Thus, we might choose to simulate patients with difficulties on specific vowels or consonants. Patient FM (Tainturier & Caramazza, 1996) for example, demonstrated exactly this sort of effect where letters were more likely to appear in the response if they were vowels than if they were consonants (71% versus 57% for six-letter words). CW (Cubelli, 1991) however, made far more errors with vowels. In cases such as CW and FM, this might be

[18] Provided by Jamie Ward (Institute of Cognitive Neuroscience - London)

explained by increasing or decreasing the threshold $T_r$ for all vowels, making them more difficult or simple to recall in the presence of lesioning. Although we included this functionality in our model, a fuller examination of how it affected error rates was deemed beyond the scope of the thesis and possibly worth considering for future work.

## 5.10 Geminates

One addition to our model that differs from the GSC model is that of a geminate production facility shown in Network A of Figure 5.2. If a letter has an active geminate status, then it will be produced twice instead of once. We argue that on similar evidence suggesting a separate CV status (e.g. Caramazza & Miceli, 1990, Miceli, et al, 2004), and a separate geminate status (e.g. Caramazza & Miceli, 1990, Tainturier & Caramazza, 1996, Miceli et al, 1995), that our architecture warrants a separate geminate production mechanism. Glasspool & Houghton (2005) use a similar approach. In order to discuss this in more detail, and to clarify our approach at arriving at an eventual model, we have delayed a more extensive discussion of this until Chapter 7.

## 5.11 Training the networks

Typically with feed forward networks, the connectionist modeller provides an input and a desired output. Through an iterative learning process, weights connecting input nodes to output nodes are modified such that correct attributes in the output are reinforced, while incorrect attributes are inhibited. Figure 5.2 shows the model as a feedforward network with many intermediary layers. In practice however, there are two distinct networks which can be trained in isolation until each demonstrates stable intact behaviour. Network B takes semantic vectors as input producing word identity vectors as output. Network A however, is trained using the unique word identities as the input on the assumption that perfect (or near perfect) values will be provided by an intact

network B. Parameters used to train each network are the same as those used with the GSC model and detailed in Appendix A.

### 5.11.1 Training Network A

The backpropagation algorithm feed inputs forward and usually considers all outputs equally important when determining how closely target and actual outputs match. The GSC model however, only requires the correct letter to be sufficiently active at the appropriate time, so activations of other letter nodes can be ignored. Letter node activations during training and recall fall into five distinct scenarios (Glasspool 1998):

1. The most active letter node corresponds to the target letter for the current position within the word, and its activation exceeds a given response threshold $T_r$.
2. The most active letter node corresponds to the target letter but its activation does not exceed $T_r$.
3. The most active letter node is not the target letter for the current position within the word.
4. The current letter position is past the end of the word, and the most active letter node does not exceed a given stop threshold $T_s$.
5. The current letter position is past the end of the word but the most active letter node exceeds $T_s$.

In scenarios one and four, the letter in its position is deemed *correct* (or irrelevant if past the end of the word), and no weight modifications are necessary. The GSC model takes corrective action rather than reinforce an already learned state. Glasspool calls this *Lazy Updating* as learning only occurs for letters involving erroneous behaviour. In a standard backpropagation error correction approach, letters having no relationship to a word's position are still inhibited causing activations to become even lower on recall. In scenarios two, and three, the target letter is reinforced, and the incorrect letter is inhibited

respectively. In scenario five, we inhibit the activation of the winning letter, since we are beyond the end of the word, and above the stop threshold. One benefit of avoiding excessive suppression of irrelevant letters is that these letters become likely candidates for erroneous production in the presence of lesioning.

Like the GSC model, we also use a *training margin*. During training (not recall), a small margin $M_t$ is added to threshold $T_r$ and subtracted from threshold $T_s$. The impact of this is to train the network with slightly more stringent thresholds than during recall. This helps to overcome the propensity of the backpropagation algorithm to learn a problem set as precisely as possible. Another benefit of the margin is that we avoid excessive errors that may result from adding noise to a very finely trained network. It is also possible in our model to use random training margins, where a margin can retain any value between two values. As discussed in Chapter 4, this innate noisiness is more physiologically plausible. The network is trained until recall is perfect for all words for 20 epochs in a row.

### 5.11.2 Training Network B

Network B is trained using backpropagation in isolation from Network A until the maximum error on any word identity is less than 1%. In order to show a frequency effect in the presence of lesioning, every high frequency word is presented on each training epoch, whereas every low frequency word is presented with a probability of 0.30. It is interesting to note that if low and high frequency words were to be presented differently in Network A, that the lazy learning algorithm would prevent a frequency effect from arising. Although Network A might have high frequency items presented more regularly than low frequency items, the network will actually stop learning high frequency items once they are properly learned (Glasspool et al, 2006).

### 5.11.3 Input Representations

To establish that our current model behaves correctly, we use the same corpora as the GSC model. There are 400 words, 100 each of length 4, 5, 6, and 7 letters selected from the MRC psycholinguistic database (Quinlan, 1993; Coltheart, 1981). Unlike Network B, Network A is expected to show no semantic or frequency effect in the presence of lesioning.

#### 5.11.3.1 Network-A Representation

For each of the 400 vectors comprising the corpus, 400 nodes are used to accommodate the word identity component of the input. Each word identity vector has four active nodes that overlap with six other vectors. Two overlap by one active node, two overlap by two nodes, and two overlap by three nodes. Vectors are randomly assigned to words.

#### 5.11.3.2 Network B Representations

For Network B, the desired output is the word identity required by Network A. As input to Network B, we use two alternative vector designs, each of which should show a semantic effect in the presence of lesioning. The first is identical to the GSC model; every word is assigned a randomly generated vector of 56 semantic features. Twenty-eight features are labelled *concrete* and 28 features are labelled *abstract*. Highly concrete words have 14 concrete features active such that every possible pair of words differs on at least five features. Low concreteness words have five abstract features active such that each pair differs by at least three attributes. High and low concreteness words thus differ in the density of their semantic representations but also in the number of active nodes. The second vector design uses a similar approach as Plaut & Shallice (1993) where concrete words have relatively more active attributes than abstract words. In the Plaut and Shallice corpus, there are 40 items, comprising 20 concrete and 20

abstract words. Of their 98-attribute vector, 31 attributes account for abstract features, and 67 for concrete features. Collectively, for all concrete words, there are 337 active concrete features, and 22 active abstract features. For all abstract words, there are no active concrete features, and 98 active abstract features. Table 5.2 summarise the probabilities of an attribute being active within each approach.

| Vector Type | Word Type | Abstract Features | Concrete Features | Totals |
|---|---|---|---|---|
| **Plaut & Shallice** | **Abstract** | 0.1597 | 0.0000 | 0.1597 |
| | **Concrete** | 0.0355 | 0.2515 | 0.2870 |
| **Glasspool** | **Abstract** | 0.0892 | 0.0000 | 0.0892 |
| | **Concrete** | 0.0000 | 0.2500 | 0.2500 |

**Table 5.2: Proportion of active attributes in semantic vectors used by the Plaut & Shallice (1993) and the GSC model.**

Our alternative vector design is therefore 98 attributes long where abstract and concrete words have the same proportion of total active attributes as described in Table 5.2 for the Plaut & Shallice design. We would therefore expect more active attributes in concrete vectors to produce more fault tolerance and a lower error rate in the presence of lesioning. A major difference between our alternative vector design, and both the Plaut & Shallice, and Glasspool designs, is that our alternative design does not define an attribute as being specifically abstract or concrete. A word's concreteness is defined purely by the proportion of active attributes.

## 5.12 Data Analysed During Recall

We validate the behaviour of each network by collating three types of comparative results. Each measures the propensity of certain types of words to be erroneous:

- *Abstract/Concrete words*

  In Networks A and B, an abstract or concrete identity is assigned to a vector. This is purely for informational purposes and does not form part of the input vector. The GSC model predicts that network A should show no significant difference between

abstract and concrete error rates, whereas in Network B, abstract words should be more susceptible to error than concrete words.

- *Low/High Frequency words*

  In Networks A and B, a low or high frequency identity is assigned to a vector, and does not form part of the input vector. The GSC model predicts that Network A should show no significant difference between the error rates of low and high frequency words, whereas in Network B, low frequency words should be more erroneous. Training by presenting high frequency items more often than low frequency items seems to reflect the performance of human subjects. This procedure has been used by at least Cohen & Servan-Schreiber (1992), Gotts & Plaut (2002) and Bullinaria (2003),

- *Simple/Complex CV words*

  This status reflects a word's orthographic composition. CV complexity does not influence how each corpus member is trained but is recorded to determine whether orthography influences a word's propensity for error in the presence of lesioning.

One possible extension to the data we current analyse may be to collate information on errors associated with specific letters. For example, is it possible to influence the model to show relatively more errors on the letter 'E' than other vowels (e.g. Kay & Hanley 1994)?

## 5.13 Quasi-subject approach

In our tests, we approach the analyses of our models by ensuring that we train and recall from networks using multiple random seeds. We expect to see qualitatively similar results across network tests as one expects to see with different patients. Mozer, Halligan & Marshall (1997) constructed an ensemble of lesioned models each slightly

different. They suggested that patients presumably have a slightly different form of deficit and an ensemble of models measures mean performance across quasi-patients, each slightly different. Inconsistent behaviour has also been shown to occur within individual patients for example by producing the same word differently on separate occasions (e.g. patient JH, Kay & Hanley, 1994, and patient AS Jonsdottir et al, 1996).

We train each network on three different random seeds. Each of these networks is then tested using five different random seeds, thus resulting in fifteen quasi-subjects. All results are averaged out to give an overall indication of the network's performance. This approach is common where a modeller seeks to minimise random skewing of results such as with ensemble networks. By testing on a number of quasi-subjects, variance across the subject-pool is minimised and representing the overall qualitative behaviour being modelled. Similar methods have been used by Hinton & Shallice (1991), Plaut & Shallice (1993), Lawrence, Giles & Fong (1996), Harm & Seidenberg (1999), Glasspool, Shallice & Cipolotti (1999), Mozer, Halligan & Marshall (1997), East (2003:26), and Monaghan & Shillcock (2003).

## *5.14 Lesioning the Networks*

As discussed in Chapter 4, a number of authors have approached lesioning by applying specific severities and showing how these compare to patient data (e.g. Hinton & Shallice, 1991, Plaut, 1995, Glasspool et al, 2006). We expand on this lesioning approach by allowing the modeller to apply multiple severities across the fifteen quasi-subjects discussed earlier. In our tests, ten distinct error severities are applied to the model. Our objective here is to ascertain whether the model's behaviour in the presence of lesioning is constrained to a limited range of damage or is robust across a broad range of severities. Severities are chosen such that at the smallest level, just over 0% of errors

on average are produced, and at the highest severity, approximately 80%-90% of errors on average are produced. Severities are generally evenly spaced across all ten values producing a progressively increasing error rate. We apply the lesion types described in Chapter 4, namely ablation, noise, scaling weights, constraining connectivity, modifying an activation function, and adding noise to an activation function.

The GSC model predicts that lesioning areas supposedly accountable for specific functions should produce predictable errors. For example, lesioning the context area in Network A is expected to affect letter order and therefore the distribution of substitutions, inserts, transpositions, and deletes. Figure 5.6 and Figure 5.7 show Networks A and B highlighting various lesion locations. As Figure 5.6 shows, Network A can be lesioned in six places: 1) Between the bias and hidden nodes, and between the bias and output nodes, 2) Between context and hidden nodes, 3) Between the word identity and hidden nodes, 4) Between the hidden and output nodes, 5) By adding noise to the letter node output activations, 6) By modifying the context function, and 7) between the bias and geminate nodes, and the hidden layer and geminate node.

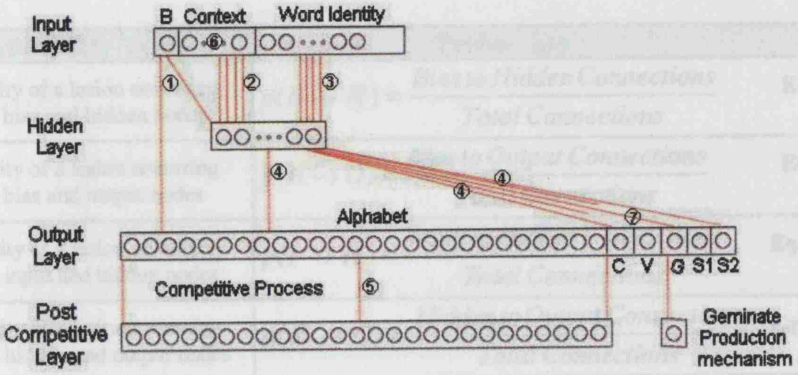



**Figure 5.6: Lesion locations within the Letter Network**

Figure 5.7 shows that Network B can be lesioned in three places: 1) Between the bias and hidden nodes, and between the bias and output nodes, 2) Between the input and hidden nodes, and 3) Between the hidden and output nodes.

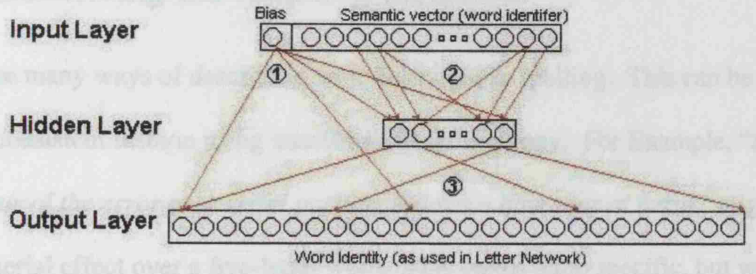



**Figure 5.7: Lesion locations within the Semantic Network**

To ensure that every connection has a proportional opportunity to affect behaviour, each is given a lesion probability relative to the total number of connections in the network. The total number of connections for each network is calculated as follows:

$$Total\ Connections = (H(I+O)) + H + O \qquad \textbf{Equation 5.7}$$

where H is the number of hidden nodes, I the number of Input nodes, and O the number of output nodes. This equation accounts for bias connections (B) to the hidden and output nodes. For Network A, CV, geminate nodes, and Stop States are considered output nodes. Therefore the probability of an error occurring in any part of the network is as follows:

| Probability Type | Probability | |
|---|---|---|
| Probability of a lesion occurring between bias and hidden nodes | $p(B \rightarrow H) = \frac{Bias\ to\ Hidden\ Connections}{Total\ Connections}$ | **Equation 5.8** |
| Probability of a lesion occurring between bias and output nodes | $p(B \rightarrow O) = \frac{Bias\ to\ Output\ Connections}{Total\ Connections}$ | **Equation 5.9** |
| Probability of a lesion occurring between input and hidden nodes | $p(I \rightarrow H) = \frac{Input\ to\ Hidden\ Connections}{Total\ Connections}$ | **Equation 5.10** |
| Probability of a lesion occurring between hidden and output nodes | $p(H \rightarrow O) = \frac{Hidden\ to\ Output\ Connections}{Total\ Connections}$ | **Equation 5.11** |

**Table 5.3: The probabilities of a lesion affecting respecting parts of the model.**

A summary of the lesion severities applied to each lesion location, and of each type is provided in Appendix D.

## 5.15 Describing the resulting behaviour

There are many ways of describing error behaviour in spelling. This can be difficult to do in a consistent fashion using unambiguous terminology. For Example, "*the behaviour of the error over serial position follows a bow shaped curve*" might describe a typical serial effect over a five-letter word. This seems quite specific, but was the curve deep, and consistent in height? Was the bow-shaped curve like a U, or like an upside-down U? Was it rounded, or pointed? Did it seem normally distributed, or was it skewed somewhat, and if so, in which direction? Undoubtedly all these features could be described using natural language, but such a description may still be open to a broad vocabulary, each word bringing its own connotations to the reader. The description may also be overly verbose making large-scale communication of the nature of the error difficult and increase the risk of inconsistency should there be a lot of data collated by different individuals. We have therefore developed a minimal vocabulary that attempts to articulate the observable behaviours described above. Ideally a simplified four or five-character representation should allow a reader to reproduce the originally observed behaviour in graphical form. There are at least two types of behaviour we are interested in analysing:

1) Serial position behaviour – How does error rate depend on the relative serial position of a letter?
2) What proportion of words is in error, and how do the letters in error relate to the words they belong to, and the letters they transpose/substitute with.

For the sake of brevity, we have deferred an extended discussion of these until Appendix B. We believe that the short hand representations are relatively concise, and portable. They are concise as they only take four to five characters to describe behaviour, and portable in the sense that they can be easily communicated and presented.

## 5.16 The scope of collated data

The literature describes many approaches to quantifying patient errors. Some authors reference single errors (e.g. Caramazza & Micelli, 1987, Jónsdóttir et al 1996), and others reference multiple errors (e.g. de Partz, 1995). We have taken advantage of modern computing power and have simultaneously collated multiple error metrics (see Table 5.4). We will therefore be able to provide comparable analyses using our model.

| Error Type | Description | Source / Reference |
|---|---|---|
| Single | Single error associated with a word | Cipolotti et al (2004) |
| Double | Two errors associated with a word | Cipolotti et al (2004) |
| Complex | More than two errors associated with a word | Cipolotti et al (2004) |
| Single87 | A single error associated with a word | Caramazza et al (1987) |
| Multiple87 | Multiple occurrences of the same error type | Caramazza et al (1987) |
| Mixed87 | Mixed occurrences of error types | Caramazza et al (1987) |
| Single90 | One or many errors of a single error type | Caramazza & Micelli (1990) |
| Mixed90 | More than one error of mixed error type | Caramazza & Micelli (1990) |
| Fragments | A resultant word length greater than 2 fewer characters than the original word length | Ward (1997) |

**Table 5.4: The metrics collated by the model and the original reference for our method of measurement.**

## 5.17 Description of letter notation

Computer-based textual output is relatively simple, offering few opportunities to convey more than the rudiments of information. We attempt to describe how the model spells a word in as informative a notation as possible. In cases where a delete may be attributed to insufficient activation above a letter threshold, for example, we will not just omit the relevant character; there is knowledge to be gained from showing what the most probable active (albeit not qualifying for production) character at that position was. Table 5.5 summarises these:

| Raw Output | Spelled Output | Interpretation of Output |
|---|---|---|
| G E N E R A L | G E N E R A L | No error is present here |
| G **[E]** N E R A L | G N E R A L | The letter 'E' has been deleted and highlighted by being embedded within square brackets. |
| C H E **E*** P | C H E E P | The letter 'E' has been doubled as a result of an active geminate status. |
| C H E **[P]** E P | C H E E P | The letter 'P' has been deleted, and followed by another letter 'E'. The double 'E' is therefore due to an embedded insert and not an active geminate |

**Table 5.5: Example model outputs, and typical errors with their interpretations.**

The first column shows the raw output by the model followed by the actual word a patient would be expected to show. The third column interprets the behaviour.

## 5.18 Scoring procedure

At a simple level, there are effectively only four types of errors: substitutions, inserts, transpositions and deletes. This broad classification of error types is however only really appropriate for words containing single errors (see Table 5.6). Distinguishing between them becomes less clear when errors of more than one type are present. In order to provide as informative an analysis as possible, we record the context of the error. Was the letter for example, substituted by another letter contained in the target word, and was the vowel substituted by another vowel or a consonant?

| Error Type | Sub Type | Example |
|---|---|---|
| Substitutions | Within word vowel | METODO → METEDO |
| | Within word consonant | METODO → METOTO |
| | Out of word vowel | METODO → METADO |
| | Out of word consonant | METODO → METOBO |
| Inserts | Within word vowel | CONTRO → CONOTRO |
| | Within word consonant | CONTRO → CRONTRO |
| | Out of word vowel | CONTRO → CONATRO |
| | Out of word consonant | CONTRO → CLONTRO |
| Deletes | Simple | ULTIMO → UL[T]IMO |
| Transpositions | Simple | ALZARE → AZLARE |
| | Exchange – two graphemes exchange place | METODO → MEDOTO |
| | Shift – One grapheme moves from one position to another | CONTRO → CRONTO |

**Table 5.6: Example single error types for substitutions, inserts, deletes, and transpositions.**

Some authors (e.g. Caramazza & Micelli 1987, Houghton et al 1994) separate shifts and exchanges. Like Kay and Hanley (1994), we chose to combine shifts and exchanges into a single classification of transposition. We discuss this in more detail further in this chapter.

## 5.19 Error types and rationales for production

In practice, an erroneous word often includes many different error types. The distinction between an exchange and a shift (Caramazza & Miceli 1990) is clear when the error involves non-adjacent letters. However, cases like ALZARE → AZLARE make it difficult to decide whether the error results from the exchange of consonants in second and third positions, or from the shift of the consonant in the third position to the second position or of the second to the third position. Although both explanations for how the error occurred are plausible and thus functionally equivalent, the scoring of each is different (see Table 5.7). Caramazza & Miceli (1990) arbitrarily consider these as exchanges, whereas we consider them to be simple transpositions of two adjacent characters. A further complication exists with transpositions such as ALTARE → ARLARE. We assume here that the L and T transpose, and the T substitutes with a within-word consonant 'R' (ALTARE → ATLARE → ARLARE). We argue that this is still a valid transposition albeit complex, rather than a delete and an addition, firstly because substitutions are more prevalent in patient data than deletes and inserts, and secondly because a single substitution is a simpler operation than a coordinated delete and insert involving the same letter position. In terms of actual outputs, a coordinated delete and insert of the correct letters is indistinguishable from a transposition.

Deletes are ostensibly exhibited in the program output by highlighting the deleted letter between square brackets STORE → STO[R]E → STOE. However, a

letter may also be *interpreted* as having been deleted. For example, in the case of STORE → STRE, there is no missing letter in position three, but the system interprets the absence of the O from the letter, and assumes a delete in the position where the O should have been. The basic scoring mechanism is outlined in Table 5.7, the foundation of which comes from Caramazza & Miceli (1990), and adapted to accommodate more complex transpositions and substitutions. Caramazza & Miceli (1990) only cater for words of single error types whereas a *forward transposition* (see Table 5.7) for example immediately assumes a double error. Therefore, although we have added extra error classifications, the assignment of points for specific errors is identical to those of Caramazza & Miceli (1990).

| | |
|---|---|
| Transpositions | An extended analysis of errors beyond single error types requires a more complex approach than used by Caramazza &Miceli 1990.<br>• Simple Transpositions<br>One point is assigned to both positions corresponding to exchanged letters E.g. MODEL → MOEDL = 1 point to positions three and four.<br>• Exchange Errors<br>One point is assigned to both positions corresponding to exchanged letters E.g. CINEMA → CIMENA = 1 point to positions three and five.<br>• Letter Shifts<br>One point is assigned to the position corresponding to the shifted letter, ½ point to the position preceding the one where the letter was moved to, and ½ point to the position following it. E.g. CONTRO → CRONTO = 1 error in position five (the original 'R' position), ½ point in position one, ½ point in position two.<br>• Complex Transpositions<br>This assumes that a transposition and substitution occur jointly. One point is credited to the position where the transposed letter now lies, and 1 point is credited to where the substitution has occurred.<br>There are two ways this can occur:<br>***Forward transposition*** –A letter transposes to the next serial position, the original position substituted by a letter other than that in the next serial position. For example, ALTARE → ARLARE shows the letter 'L' transposed forward, and the letter 'T' substituted by 'R'. Here, a transposition is assigned to positions two and three, and a substitution to position two.<br>***Backward Transposition*** - A letter transposes to the previous serial position, the original position substituted by a letter other than that in the previous serial position. For example, CONTROL → CONRPOL shows the letter 'R' transposed backwards, and the letter 'T' substituted by 'P'. Here, a transposition is assigned to positions four and five, and a substitution to position five.<br>Note that while transpositions may have more than one point credited to various serial positions. The transposition itself is considered a single error. A transposition occurring with a concomitant substitution will by definition be a double error. |
| Insertions | A ½ point is assigned to the position before and after the inserted letter. E.g. FOGLIA → FLOGLIA = ½ point in position one and ½ point in position two. If the letter inserted is deemed to be beyond the end of the original word, a single point is assigned to that inserted position. E.g. FOGLIA → FOGLIAL would have 1 point assigned to position seven. |
| Deletes | A single point is assigned to the position where the delete occurred. E.g. ULTIMO → UL[T]IMO → ULIMO = 1 error in position three.<br>In both cases above, a delete would be assigned to position four. |
| Substitutions | A single point is assigned to the position where the substitution is noted. E.g. SECOLO → SELOLO = 1 error in position three.<br>Where a complex transposition presents itself, the credit for the substitution lies in the position where the transposed letter *should* have been. E.g. ALTARE → ARLARE. The credit for the substitution lies in position two. |

**Table 5.7: Scoring mechanisms assigned to each error type, and associated position. This is an extension of the approach used by Caramazza & Miceli (1990).**

## 5.20 Measurement of Positional Errors

The corpora used in this study comprise words of various lengths and in some cases, generate errors beyond a word's natural length due to inserts, or additions at the end of the word. In order to analyse error patterns across words of various sizes, we use a modification (Shallice, personal communication) of the oft-referenced Wing & Baddeley (1980) approach, which is expanded on in Appendix B. The Wing & Baddeley approach is typically used to normalise errors across five standard serial positions. Table 5.8 shows how they achieve this in a general format, and Table 5.9 shows its application to words of three to seven characters.

| Total Elements | Region | | | | |
|---|---|---|---|---|---|
| | A | B | C | D | E |
| k*5 + 1 | k | k | k+1 | k | k |
| k*5 + 2 | k+1 | k | k | k | k+1 |
| k*5 + 3 | k+1 | k | k+1 | k | k+1 |
| k*5 + 4 | k+1 | k+1 | k | k+1 | k+1 |
| (k+1) * 5 | k+1 | k+1 | k+1 | k+1 | k+1 |

**Table 5.8: Wing and Baddeley (1980) table showing relative position in a five-region format**

The cells in the section titled **Region** describe how many items can be attributed to each position depending on the word length. Therefore, a four letter word, would have an error in letter position one attributed to region A, an error in letter position two to region B, an error in letter position three to region D, and an error in letter position four in region E.

Caramazza et al. (1987) use the same Wing and Baddeley scoring mechanism as means of providing a uniform score across different word lengths. It should be noted that they omit four-letter words when collating their results.

| Word Length | A | B | C | D | E |
|---|---|---|---|---|---|
| | Original letters to use in these positions | | | | |
| 3 | 1 | | 2 | | 3 |
| 4 | 1 | 2 | | 3 | 4 |
| 5 | 1 | 2 | 3 | 4 | 5 |
| 6 | 1 | 2 | 3, 4 | 5 | 6 |
| 7 | 1, 2 | 3 | 4 | 5 | 6, 7 |
| 8 | 1, 2 | 3 | 4, 5 | 6 | 7, 8 |

**Table 5.9: The Wing and Baddeley (1980) approach applied to errors in words of various lengths.**

A closer look at Table 5.9 shows that four letter words can never generate errors in region C, meaning that an investigation of purely four letter words would never provide a bow-shaped error curve. This appears to be an inherent fault in the W&B mechanism. Indeed, including many four-letter words in a corpus containing various word lengths may inappropriately skew the results such that the expected bow-shaped serial position curve is not produced. It may be that this feature was sufficient to warrant Caramazza et al (1990) to exclude four letter words from their analyses. Of more interest is the fact that six letter words also skew the results giving overt importance to position three, and accentuating the expected bow-shaped error distribution. Caramazza et al focused solely on six letter words in their study. An alternative and arguably more balanced method of error normalisation is described in more detail in Appendix B.

## 5.21 Conclusion

In this chapter we examined the core structure of the GSC model. Our primary motivation was to determine whether the original model satisfied the functional requirements of a more comprehensive model. Our secondary motivation was to extend the range of possible analyses using existing patient data as a guide. In summary, we believe our additions to the model include:

1. *Multiple context types* – Specifically we will compare the behaviour of two control mechanisms promoting errors in medial serial positions with a third that should not.
2. *Refined inhibitory mechanism* – We believe that our approach is more in line with the theoretical boundaries of the sigmoid activation function
3. *Identification of an extra threshold* – The unknown threshold can be said to reflect the behaviour of patients who leave obvious blank spaces where letters should be. Although we do not test this further, we felt it worth highlighting as an area that fits naturally with the existing GSC approach.
4. *Adding a geminate facility* – This will compensate for the GSC model not currently being able to learn words containing double letters. We expand on this further in Chapter 7.
5. *Examination of other semantic designs* – By exposing the model to alternative semantic vectors, we will be able to ascertain how much of the semantic behaviour claimed by Glasspool et al relies on their original semantic design.
6. *Provision of an orthographic comparison* – The new model can ascertain whether words of simple or complex orthography show significantly different error rates.
7. *Introduction of quasi-subject data* – By using fifteen quasi-networks and averaging across all results, we intend to substantiate the robustness of the model by mitigating critique that results may be affected by selective starting seeds or lesion location.
8. *Applying multiple lesion severities* – Averaging across multiple lesion severities should expose the model to more rigorous testing than originally shown in the GSC model. The new model allows damage to be applied in many more

locations, and in a methodical fashion, again to avoid critique of a prescriptive lesion strategy.

9. *Introduction of a short descriptive notation* – Long-form descriptions of spelling behaviour can be ambiguous and inconsistent. We aim to reduce the ambiguity across a variety of spelling behaviours making the communication of this easier.
10. *Provision of multiple error categories* – The literature shows multiple means of describing errors. The new model collates a number of these allowing the same results to be viewed in a variety of ways.
11. *More transparency in processing* – In examining spelling errors, we expose the mechanisms used by the model by showing information normally *hidden* or missing in patient data. This allows us to question why the model behaves as it does.
12. *Refined scoring* – We expanded on the original approach by Caramazza & Miceli (1990) to take into consideration more complex error types.
13. *Refined error normalisation* – In this chapter we critiqued aspects of the oft-cited Wing & Baddeley (1980) normalisation method. We use an alternative procedure that we claim is a fairer balance of errors across five standard positions.

As we examined in Chapter 3, investigations by Glasspool et al (2006) seemed to provide an appropriate explanation for both type-A and type-B GBD symptoms. The extensions to our model in addition to the more rigorous analyses will allow us to investigate their claims further. In the next chapter we examine the behaviour of the augmented model in more detail making direct comparisons to symptoms of GBD and DD exhibited by patients

# 6 Analysis of Results

## 6.1 Introduction

In Chapter 3, we examined the relationship between aspects of graphemic buffer disorder (GBD), and deep dysgraphia (DD) as each seemed to exhibit some common behavioural characteristics. Work by Glasspool, Shallice & Cipolotti (2006) has been shown to explain aspects of both syndromes, and Chapter 5, described extensions to their work, which for the sake of brevity we called the 'GSC model'. In Chapter 4 we described a number of connectionist lesion types inspired by damage to biological systems and we will use these techniques to damage our model noting behaviour comparable to properties of GBD and DD. One aim of the GSC model as described by Glasspool et al was to determine whether a modular network would be able to explain variant (*type-A* and *type-B*) GBD symptoms by independently lesioning distinct sub-networks, each responsible for the source of the GBD behaviour.

We also investigate further the effect of the CV nodes discussed in Chapter 5. If words are simply linearly ordered strings, and CV nodes have minimal influence, then omitting these nodes should show no noticeable loss of a CV effect. In other words, consonants and vowels should have no propensity to transpose or substitute with other consonants and vowels respectively. For brevity through the rest of the thesis, we will consider consonants and vowels as belonging to separate *classes*. An example of a *within class* substitution is a consonant replaced by another consonant. An example of an *out of class* substitution is a vowel replaced by a consonant. There is also evidence in the literature a) that orthographic structure may influence error behaviour (e.g. Caramazza & Miceli, 1990, Tainturier & Caramazza, 1996) as well as counter views (Jonsdottir et al, 1996, Kay & Hanley, 2004), and b) that a minimum complexity

principle may influence resultant word structure in the presence of lesioning (e.g. Caramazza & Miceli, 1990, Kay & Hanley, 1994). We therefore investigate whether the model shows complex CV words to be more error-prone than simple CV words, and introduce a CV-complexity quotient giving an empirical measure of orthographic structure before and after damage. This quotient allows us to investigate whether the model (like patients) shows evidence of a minimum complexity principle.

In Chapter 5, we described three serial context mechanisms assisting the sequential ordering of a word's constituent letters - Moving Window (Burgess & Hitch, 1992), Initiator/End or IE (Houghton, 1990), and Positional. The first two have been examined by different authors for explaining serial errors relative compared to GBD data. Throughout this chapter, we will examine their relative performance to the third context mechanism, which is not expected to produce plausible GBD effects. Serial context is intrinsic to the network input, so we investigate how each context type affects error distribution, and errors associated with concreteness and frequency. We also examine the model's ability to simulate selective properties of another disorder not discussed by Glasspool et al (but see Ward & Romani, 1998), namely neglect. A further extension to the model includes simulating geminate related behaviour but we leave a fuller investigation of this until chapter 7.

## *6.2 Description of patient behaviours being modelled*

We will first revisit the properties of each disorder we intend to simulate.

### 6.2.1 Graphemic Buffer Disorder

The literature (e.g. Posteraro et al, 1988, Cubelli et al, 1991, Miceli et al, 1985, Caramazza et al, 1987, Caramazza et al, 1990, Hillis & Caramazza, 1989, Jonsdottir et

al, 1996, Kay & Hanley, 1994, McCloskey et al, 1994, Tainturier & Rapp, 2004) lists a number of GBD properties:

1) Spelling errors should tend to increase as a function of word length, and are typified by the presence of substitutions, transpositions, deletes and additions, or any combination of these.
2) Spelling accuracy is unaffected by factors such as word frequency, concreteness, grammatical class (verb, noun, function words), and lexical status. A consonant/vowel (CV) effect has also been noted in GBD patients manifested by CV preservation in transpositions and substitutions.
3) Serial position of the errors will be reflected by a bow-shaped curve

### 6.2.2 Deep Dysgraphia

Deep dysgraphia patients are assumed to have damage prior to the graphemic buffer and thus produce different symptoms:

1) They are expected to exhibit an inability to spell non-words with words relatively spared (e.g. Nolan & Caramazza, 1983, Tainturier & Caramazza, 1996, Schiller et al, 2001). Like the GSC model however, our model only works with known lexical entries.
2) Deep Dysgraphic patients usually also produce semantic errors such as TIME → CLOCK (Bub & Kertesz, 1982), GERMANY → SWEDEN, ARM → LEG, (Cipolotti et al, 2004). Theoretically our model could produce this effect, but the ability to do so is influenced crucially by the choice of semantic vectors used in the semantic system. As discussed later in this chapter, our corpora are not designed with semantically similar corpus members being allocated similar feature vectors, so such an effect cannot be simulated. In the discussion at the end of this chapter, we

describe tests using a corpus where semantically similar words are allocated similar vectors, and the model does indeed produce semantic errors.

3) Deep dysgraphic patients tend to make more errors on abstract than concrete words (e.g. Bub & Kertesz 1982, Nolan & Caramazza, 1983, Cipolotti & Warrington, 1996, Schiller et al, 2001); this is also shown by the GSC model.
4) Unlike GBD patients who produce a classic bow-shaped serial position curve, some DD patients produce serially increasing errors as a function of word length (e.g. Schiller et al, 2001, Ward & Romani, 1998, Katz, 1991).

### 6.2.3 Neglect

A predictable symptom of GBD patients is the bow shaped error curve, yet one disorder known to demonstrate different serial position curves is unilateral neglect; a collection of behavioural symptoms in which patients appear to ignore, forget or turn away from contralesional space (Heilman et al, 1983). The effect is usually contralesional, yet Baxter & Warrington (1983) describe a patient ORF showing right side neglect in writing due to a right brain lesion. Errors in neglect tend to increase with letter position, and usually increase serially from left to right, or right to left depending on the nature of the neglect. Ward et al (1998) simulated an increasing serial position curve using mechanisms similar to the GSC model, and Glasspool et al (1999) examined the GSC model's ability to simulate errors affecting the first serial positions as shown by patient CMB. Despite this, the GSC model has so far been unable to account for left side neglect symptoms where one might expect fewer errors as a function of serial position, affecting the entire word. Our coverage of neglect in this chapter is solely to situate the relevance of the *context* described in Chapter 5, and determining whether disorders besides GBD and DD can be explained by such mechanisms.

## 6.3 Experimental Method

### 6.3.1 Approach to testing

In Chapters 4 and 5, we proposed a method of training and testing networks with multiple random seeds. This provides multiple quasi-patients, minimising variance between results while retaining the qualitative nature of the overall test. Each network is trained using three random seeds, and then lesioned and tested with five random seeds, providing us with fifteen data sets for each lesion location and lesion type.

### 6.3.2 Corpus Characteristics

The model is exposed to three corpora to determine how robust it is with respect to different input data sets. Each corpus contains the same 400 words used by Glasspool et al (2006). The Graphemic Buffer, and Semantic System networks, known as network A, and B respectively use different feature vector sizes based on the length of each network's input layer. We expand on Glasspool et al's original corpus design using various feature vectors. 1) Network A uses the same vectors as Glasspool et al. 2) Corpus B-DG uses the same vectors for network B as Glasspool et al. 3) Corpus B-PS uses an alternative vector design for network B based on a similar approach to Plaut & Shallice (1993). As mentioned earlier, semantically similar corpus members do not necessarily have similar semantic feature vectors, so the model is not expected to produce semantic errors. Rather, the network should more accurately model the propensity for abstract words to be more erroneous than concrete words and we expand on this further towards the end of the chapter.

### 6.3.3 Experimentation

For the purposes of the thesis, we define an experiment as the application of a specific lesion type and severity to a defined location (focal or diffuse) and to a network trained with a given corpus. Table 6.1 describes the lesion locations in each network and lesion types described in Chapter 4, which are applied to each.

| Network | Specific Lesion Location | Applicable Lesion Types |
|---|---|---|
| Graphemic Buffer (network A) | Between bias and hidden nodes and bias and output nodes | Ablation, Noise, Constraint, Scaling |
| | Between context and hidden nodes | |
| | Between word-identity and hidden nodes | |
| | Between hidden and output nodes | |
| | Between bias and geminate nodes and hidden later and geminate node | |
| | Adding noise to the letter output activations | Noise |
| | Context function | Modified I and E functions |
| Semantic System (network B) | Between the bias and hidden noses and between bias and output nodes. | Ablation, Noise, Constraint, Scaling |
| | Between the input and hidden nodes. | |
| | Between the hidden and output nodes. | |

**Table 6.1 : Lesion locations and types applied to each location for both sub-networks. We expand on our modified I and E functions toward the end of this chapter.**

Table 6.2 introduces the seven experiments used in our study, each designed to investigate one or more aspects of the model's architecture, or the nature of the data being used.

| Title | Description |
|---|---|
| **A-MW** | Tests network A using a ***moving window*** context (e.g. Burgess & Hitch, 1992, see 5.6.2.2.2). Aims to reproduce GSC results for symptoms of GBD. |
| **A-IE** | Tests network A using the ***Initiator/End*** context (Houghton, 1990, see 5.6.2.2.3) comparing behaviour to the moving window and positional contexts. |
| **A-POS** | Tests network A using the ***positional*** context (see 5.6.2.2.1), comparing behaviour to the moving window and IE contexts. |
| **A-NOCV** | Tests network A using a ***moving window*** context, and trained without CV nodes thus clarifying the influence of CV nodes. |
| **THRESHOLD** | Tests network A using a ***moving window*** context to highlight the effect of modifying the letter presentation threshold (see Chapter 5) |
| **B-DG** | Tests network B using the semantic vector design of Glasspool et al (2006) to reproduce their results for symptoms of DD. |
| **B-PS** | Tests network B using a modified vector design based on that described by Plaut & Shallice (1993). |

**Table 6.2 : Experiments used to analyse the nature of our results. For more detail on context types and where each network is situated in the model, see Chapter 5.**

Note that Table 6.2 references no geminate related tests. In Chapter 7, we investigate geminate behaviour in substantially more detail and thus leave a fuller examination of related corpora, and lesioning until then.

### 6.3.4 Presentation of Results

Throughout this chapter, we will present our results using the short descriptive notations described in Appendix B. We apply a number of lesion types of progressive severities to various locations in order to analyse behavioural aspects of the network compared to some available patient data. All table values are averages of single or total errors across all word lengths. Further, the values presented in serial position analyses are averages across all lesion severities, and use a modified scoring mechanism discussed in Chapter 5 based on one originally described by Caramazza & Miceli (1990). In addition, we use a modified method of balancing error accountability across five serial positions rather than the oft-cited Wing & Baddeley (1980) method, also described in Appendix B. The literature shows various examples of tables of patient results showing either number of correct letters/words (e.g. Kay & Hanley, 1994, Cipolotti et al, 2004), or number of letters/words in error (e.g. Ward, Olsen & Romani, 1998, Tainturier & Rapp, 2004, Orpwod & Warrington, 1995). As the thesis focuses on the effects of damage, we present results measured by number of words/letters in error. To simplify the presentation of statistical significance in tables, we use the symbols outlined in Table 6.3 rather than list individual significance values (e.g. $p < 0.023$).

| Level of Confidence | Symbol |
|---|---|
| $p < 0.05$ | * |
| $p < 0.01$ | ** |
| $p < 0.001$ | *** |

**Table 6.3: Symbol representations of confidence levels. These are two-tail significances.**

Through the chapter, we also make use of $\chi^2$. This measure may be inappropriate if any expected frequency is less than 1.0 or if the expected frequency is less than 5.0 in more than 20% of cells. In a 2x2 case of an *unsafe* $\chi^2$ test a refinement to the measure known as Yates' correction will be used. In a 3x2 case of an unsafe $\chi^2$ test, we will use the Fisher Exact probability test. All of these tests are non-directional (two-tailed).

### 6.3.5 The Testing Environment

To cater for large data volumes and, in computer science terms *"complex query"* types we developed a testing and analysis workbench ensuring a level of consistency in defining experiments, and allowing us to subject the collated data to various analyses. The workbench is composed of two main programs. The first, *GBDS* (Graphemic Buffer Disorder Simulator) is programmed in ANSI C and used to train, lesion, and test our experiments. It is written so as to allow flexible definition of run-time parameters (see Appendix G). As there is no need to recompile our program for subtly different tasks, we minimise the risk of programmatic errors in reproducing our results. The second program *AnalyseApp* is written in Microsoft Visual Basic and used to define experiments, view our results in tabular and graphical formats, and create scripts for use by GBDS. Results are stored in a relational database to cater for large quantities of data, and for a range of analytics using industry-standard reporting tools should this be of interest at a future date.

### 6.3.6 Establishing significance

In the rest of this chapter we evaluate whether lesioning tends to produce more abstract than concrete errors, more errors on low frequency than high frequency words, and whether orthographic complexity affects error rates. The key question we are asking is "are the distributions of the two error curves *sufficiently* different to be considered

significant?". This is a straight forward question, but not answered easily due to the nature of the errors and lesion strategy. Firstly, our raw data comprises 150 pairs of comparative errors; fifteen pairs at ten progressively increasing lesion severities (see Appendix E for an example). It is not enough to determine whether abstract and concrete errors seem to be from different distributions, they should be substantially different as well. One might suppose that where two error rates are significantly different, they should be noticeable different as well, but this is not always the case. For example, a Wilcoxon test will determine that abstract errors which are on average 0.00001 higher than concrete errors for ten progressive lesion severities will be significantly different ($p < 0.005$). It is therefore useful to augment the significance comparison with a further test of magnitude difference between error curves to determine how substantial the differences are. A typical comparison between error rates across lesion severities will look similar to the curves in Figure 6.1. Note that although Figure 6.1 (a) seems to show very similar error rates, and Figure 6.1 (b) shows very different error rates, a Wilcoxon test considers both of these significantly different.

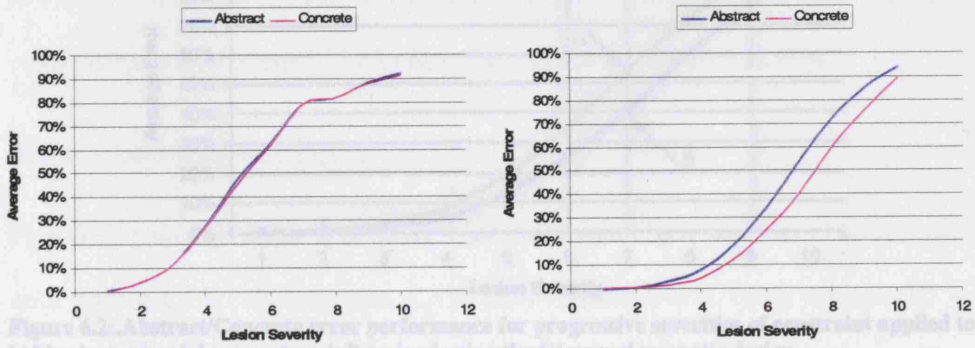



**Figure 6.1: Two instances of abstract and concrete single errors considered significant using Wilcoxon. (a) constraint applied from the input layer to hidden nodes in Network A trained with the IE context, and (b) constraint applied from hidden nodes to output nodes in network B.**

The overall magnitude of the difference between comparative measures (abstract and concrete, low and high, and complex and simple) is calculated using the following:

$$m = \frac{\sum(\beta - \alpha)}{N}$$ **Equation 6.1**

where $m$ is the magnitude of the difference between comparative values, $N$ is the number of comparative pairs, and $\alpha$ and $\beta$ represent comparative items from Table 6.4.

| $\alpha$ | $\beta$ |
|---|---|
| Abstract words | Concrete words |
| Low frequency words | High frequency words |
| Complex CV orthography | Simple CV orthography |

**Table 6.4 : Meanings of $\alpha$ and $\beta$ describing comparative values**

The sign of the magnitude difference indicates whether there is a general tendency for errors to be more prevalent on abstract or concrete, low or high frequency, or complex or simple CV words. A negative magnitude for example indicates a propensity for words of the type in the $\alpha$ column to be in error. We assume that where the absolute value of the magnitude exceeds 2.5%, then the difference between comparative values is considered *substantial* (as opposed to significant).

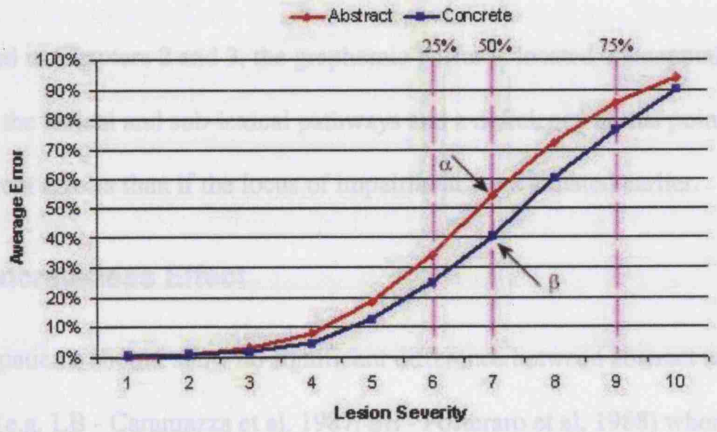



**Figure 6.2:.Abstract/Concrete error performance for progressive severities of constraint applied to hidden/output weights in network B trained using the Glasspool semantic design.**

In addition to an average magnitude difference we also provide an indication of how the average error changes across lesion severity. We first identify those severities where the average of $\alpha$ and $\beta$ errors are closest to 25%, 50%, and 75%. For these three severities, the values of $\beta - \alpha$, should provide an overall indication of how the error

difference behaves after removing floor and ceiling effects. We demonstrate this more clearly using Table 6.5, which is based on the error curves of Figure 6.2.

Identifying our three indicative values is an exercise in clarifying the trend between average errors and each of 25%, 50%, and 75%. In Figure 6.2, it occurs at severities six, seven, and nine. We then calculate the abstract and concrete differences at these points.

| Severity | Abstract | Concrete | Average | $\beta - \alpha$ |
|---|---|---|---|---|
| 1 | 0.13% | 0.50% | 0.32% | |
| 2 | 0.83% | 0.77% | 0.80% | |
| 3 | 3.40% | 1.90% | 2.65% | |
| 4 | 8.33% | 4.67% | 6.50% | |
| 5 | 18.63% | 12.77% | 15.70% | |
| 6 | 34.33% | 25.33% | 29.83% | (25%) -9.00% |
| 7 | 54.37% | 40.77% | 47.57% | (50%) -13.60% |
| 8 | 72.30% | 60.43% | 66.37% | |
| 9 | 85.40% | 76.03% | 80.72% | (75%) -9.37% |
| 10 | 94.20% | 89.73% | 91.97% | |

**Table 6.5: Average abstract and concrete total errors by lesion severity. The β-α column highlights where the average of abstract and concrete errors approximate 25%, 50%, and 75%.**

## 6.4 Expected Results

As described in Chapters 2 and 3, the graphemic buffer is located conceptually at the junction of the lexical and sub-lexical pathways and a deficiency at this point would show different effects than if the locus of impairment were situated earlier.

### 6.4.1 Concreteness Effect

Pure GBD patients should show no significant difference between abstract and concrete error rates (e.g. LB - Caramazza et al, 1987, SE - Posteraro et al, 1988) whereas DD patients are expected to produce more errors on abstract than concrete words (e.g. VS - Nolan & Caramazza, 1983, JC - Bub and Kertesz, 1982). Table 6.6 compares abstract and concrete error rates after lesioning the model. The first column shows the lesion location as described in Chapter 5 and the second column describes the associated lesion type described in Chapter 4. The columns, A-MW, A-IE, and A-POS refer to damage to

the Graphemic Buffer network (network A) where the model was trained using the moving window, Initiator/End, and positional contexts respectively. The columns B-DG, and B-PS refer to lesioning applied to the Semantic System (network B) where the model was trained using the original Glasspool et al semantic vector design, and the Plaut & Shallice approach respectively. The sub-column titled *'Sig. m'* shows the Wilcoxon significance between abstract and concrete errors followed by the magnitude of the average difference between error rates; a magnitude with a negative sign indicates more abstract than concrete errors. Substantial magnitude differences greater than ±2.5% are highlighted in yellow or green. The sub-column titled *Quartiles* describes the difference between error rates at the three locations where the average error rates differ by approximately 25%, 50%, and 75% respectively indicating how error rates behave across lesion severity.

Our first overall observation is that networks A and B behave differently with respect to concreteness. In Table 6.6, comparing the number of significant substantial differences across network types (5/63, and 19/24) showed a significant difference ($\chi^2(1) = 44.14$, $p < 0.001$). This suggests that the model behaves in the hypothesised fashion where patients with damage to the graphemic buffer produce a negligible concreteness effect, and those with damage to the semantic system do show an effect. In the model, these effects are reflected by damage to network A and B respectively.

| Lesion Location | Lesion Type | A-MW | | A-IE | | A-POS | | B-DG | | B-PS | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles |
| Random | Ablate | * 1.1 | 1.7, 2.2, 1.8 | ** -1.3 | -1.3, -2.1, -1.6 | * 0.9 | 1.1, 0.0, 1.1 | ** 9.3 | 10.1, 14.6, 12.9 | * -1.6 | 2.0, -2.2, -2.7 |
| | Noise | n.s. 0.7 | 0.8, 0.9, 1.8 | n.s. -0.6 | -2.1, -0.4, -0.6 | * 1.0 | 1.5, 2.7, 1.8 | n.s. -0.5 | 0.6, -1.3, -2.5 | ** -2.0 | -1.6, -1.7, -2.8 |
| | Constrain | ** 3.0 | 2.3, 6.3, 4.3 | n.s. -0.3 | -0.5, 0.3, 0.5 | n.s. -0.7 | 0.2, -0.1, -1.3 | ** 7.8 | 10.1, 9.4, 8.1 | **-12.5 | -11.7, -20.5, -19.3 |
| | Scale | * 2.6 | 6.3, 4.7, 5.5 | n.s. 0.2 | 0.3, -0.2, 0.8 | n.s. -0.4 | 1.7, -1.7, -3.5 | ** -8.8 | -9.5, -13.7, -11.8 | ** -29.1 | -44.0, -63.3, -63.3 |
| Input to Hidden | Ablate | n.s. 0.6 | -1.0, 0.0, 0.7 | n.s. -0.2 | -1.2, -0.6, 1.0 | n.s. -0.3 | -0.1, 0.0, -1.2 | ** 11.6 | 9.3, 14.7, 8.8 | * -2.2 | 1,0, -0.1, -5.5 |
| | Noise | * 0.8 | 1.8, 1.3, -0.8 | n.s. -0.1 | 0.8, 0.2, -0.8 | n.s. 0.7 | 0.6, -2.0, 0.9 | ** 16.3 | 20.1, 26.6, 17.0 | * -5.8 | -3.8, -7.7, -10.1 |
| | Constrain | ** 2.5 | 1.5, 5.2, 4.0 | * -0.5 | -0.3, -2.6, -0.2 | n.s.-0.5 | -0.6, 0.3, -1.1 | ** 11.3 | 12.3, 13.2, 12.9 | ** -20.5 | -20.0, -31.0, -33.4 |
| | Scale | ** 3.0 | 2.2, 6.7, 4.2 | n.s. -0.6 | -1.2, 0.0, 0.3 | n.s. -0.8 | 1.2, 1.0, -1.5 | ** -13.7 | -14.7, -21.0, -17.0 | ** -32.1 | -53.0, -72.0, -72.0 |
| Context to Hidden | Ablate | * 0.8 | 2.3, 0.8, 1.3 | n.s. -0.2 | -0.4, 0.1, 0.0 | n.s. 0.6 | 0.0, -0.3, 1.5 | N/A | | | |
| | Noise | ** 1.1 | 2.2, 2.3, 0.7 | n.s. 0.3 | 0.0, 0.0, -0.8 | n.s. 0.3 | -0.6, -0.1, -1.2 | | | | |
| | Constrain | ** 1.4 | 2.0, 3.6, 0.6 | * -1.1 | -0.6, -1.8, -5.4 | * -1.0 | -1.5, -1.3, -0.9 | | | | |
| | Scale | n.s. 0.7 | 3.7, 3.0, 1.2 | n.s. -2.1 | 0.8, -5.8, -6.3 | n.s. -0.4 | -1.8, 1.3, 1.0 | | | | |
| Word Identity to Hidden | Ablate | ** 0.8 | 0.0, 0.6, 0.6 | * -0.6 | -0.2, 0.0, -1.1 | n.s. 0.2 | 1.8, 1.2, 0.7 | | | | |
| | Noise | n.s. 0.0 | -0.4, 0.2, -0.6 | n.s. -0.3 | -0.1, -2.5, -0.5 | n.s. 0.5 | -0.2, -1.6, 0.9 | | | | |
| | Constrain | * 1.6 | 3.3, 4.8, -0.3 | * 0.7 | -0.3, 1.5, 0.6 | * 0.7 | 1.1, 1.6, -0.3 | | | | |
| | Scale | n.s. 1.5 | 6.2, 4.8, -1.8 | ** 1.3 | 1.8, 0.3, 2.0 | n.s. 0.4 | -0.2, 2.3, -1.3 | | | | |
| Hidden to Output | Ablate | ** 1.7 | 1.2, 2.2, 2.7 | n.s. -0.2 | -0.2, -1.1, 0.8 | * 0.6 | 0.2, 1.5, 1.7 | ** -11.3 | -7.4, -13.4, -13.7 | ** -7.5 | -5.5, -10.0, -8.6 |
| | Noise | * 0.7 | -0.1, 2.7, 0.9 | n.s. -0.6 | -1.9, 2.7, -1.1 | * 0.5 | -0.8, -0.1, 0.7 | ** -14.7 | -13.8, -20.9, -15.3 | ** -1.9 | -5.7, -2.2, -1.6 |
| | Constrain | n.s. 0.8 | 1.4, 3.5, 1.3 | n.s. -0.2 | -1.3, 0.0, 1.7 | n.s. 0.2 | 0.6, -0.8, 0.3 | ** -5.9 | -9.0, -13.6, -9.4 | ** -4.1 | -6.6, -9.0, -5.4 |
| | Scale | ** 3.4 | 3.5, 3.2, 2.5 | n.s. 0.4 | -1.2, 1.7, 2.7 | n.s. 1.4 | 3.2, -1.7, 1.5 | ** 5.9 | 10.2, 11.2, 4.5 | ** -3.9 | -2.7, -7.0, -5.7 |
| Output | Noise | ** 1.2 | 1.9, 1.3, 1.2 | * -0.9 | -1.2, -0.8, -0.2 | * 1.3 | 0.2, 2.6, 1.4 | N/A | | | |
| **Average magnitude** | | **1.4** | | **-0.3** | | **0.2** | | **0.6** | | **-10.3** | |
| **Significant and m≥ ±2.5** | | **5/21** | | **0/21** | | **0/21** | | **11/12** | | **8/12** | |

**Table 6.6: Abstract and concrete error comparisons across lesion types, locations, and severities for total errors. Positive magnitudes indicate more concrete errors and negative magnitudes indicate more abstract errors. See previous page for a description of each column.**

Our first observation regarding network A is that the type of serial context (A-MW=5/21, A-IE=0/21, and A-POS=0/21) does have an effect on concreteness (Fisher's Exact, $p = 0.008$) and that the moving window (A-MW) context may be more prone to error across as broad a range of lesion severities than the IE (A-IE) and positional (A-POS) serial contexts are, and constrain and scale lesions usually produce this effect. Both lesion types reduce overall weight contributions to hidden and output-layer nodes such that activation functions are less able to produce meaningful inputs. The effect of the moving window and IE contexts is to provide relatively more errors in medial serial positions, and a consequence of this medial instability may be that the network is more susceptible to lower lesion severities. We believe this can be explained on the basis that lesioning adds a level of instability to all word positions, and when combined with the innate instability associated with medial letters more errors are inevitable. It is possible therefore, that constrain and scale lesions exceed a critical point of stability at which the network can function properly. Note that the positional context does not affect the robustness of serial positions at all, and returns the least average magnitude.

At a superficial level, it might appear that semantic vector design is not a factor producing a concreteness effect in network B. After all, the B-DG (11/12) and the B-PS designs (8/12) are not significantly different (Yates' $\chi^2(1) = 1.011$, $p > 0.31$). However, this information needs to be qualified by the fact that the B-DG design only returned five values with a negative magnitude (highlighted in yellow). In other words, six lesion locations (highlighted in green) produced substantially more concrete errors than abstract errors, which is surprising and behaves contrary to patient behaviour. The B-PS design returned all negative magnitudes. In summary then, design choice does not affect significance, and magnitude, but does affect the direction of the difference, which is absolutely crucial.

For the sake of brevity, we provide a more comprehensive examination of the underlying dynamics of the semantic network using each design in Appendix H. In summary however, the B-DG design is very prescriptive about which input nodes are used for abstract and concrete words. Of the 56 attributes, 28 are labelled *concrete* and 28 *abstract*. Concrete words use more active features (fourteen) than abstract words (five), yet only use half of the input vector. This is more apparent from the two sample networks at the top of Figure 6.3 demonstrating the typical approach taken by both designs. Below each is a histogram showing the sum of active (i.e. ON) features across all corpus members. The Plaut & Shallice approach clearly provides a more balanced input reflected by less erratic behaviour in the presence of damage. With the Glasspool design, the relatively low input provided by abstract words is compensated for by the hidden nodes being active for many words. Under the right conditions, abstract words may therefore be less susceptible to damage producing more errors in concrete words. This is also exacerbated by the fact that weights from the left-most (abstract) nodes have much stronger weights than from those on the right. In the presence of damage, the limited input from these nodes multiplied by strong connectivity may also assist abstract words to be recalled correctly. This is covered in more detail in Appendix H.

We should also clarify the plausibility of network B's connectionist design. It was clear after investigating the original Glasspool et al vector design that there may be possible theoretical issues associated with it.

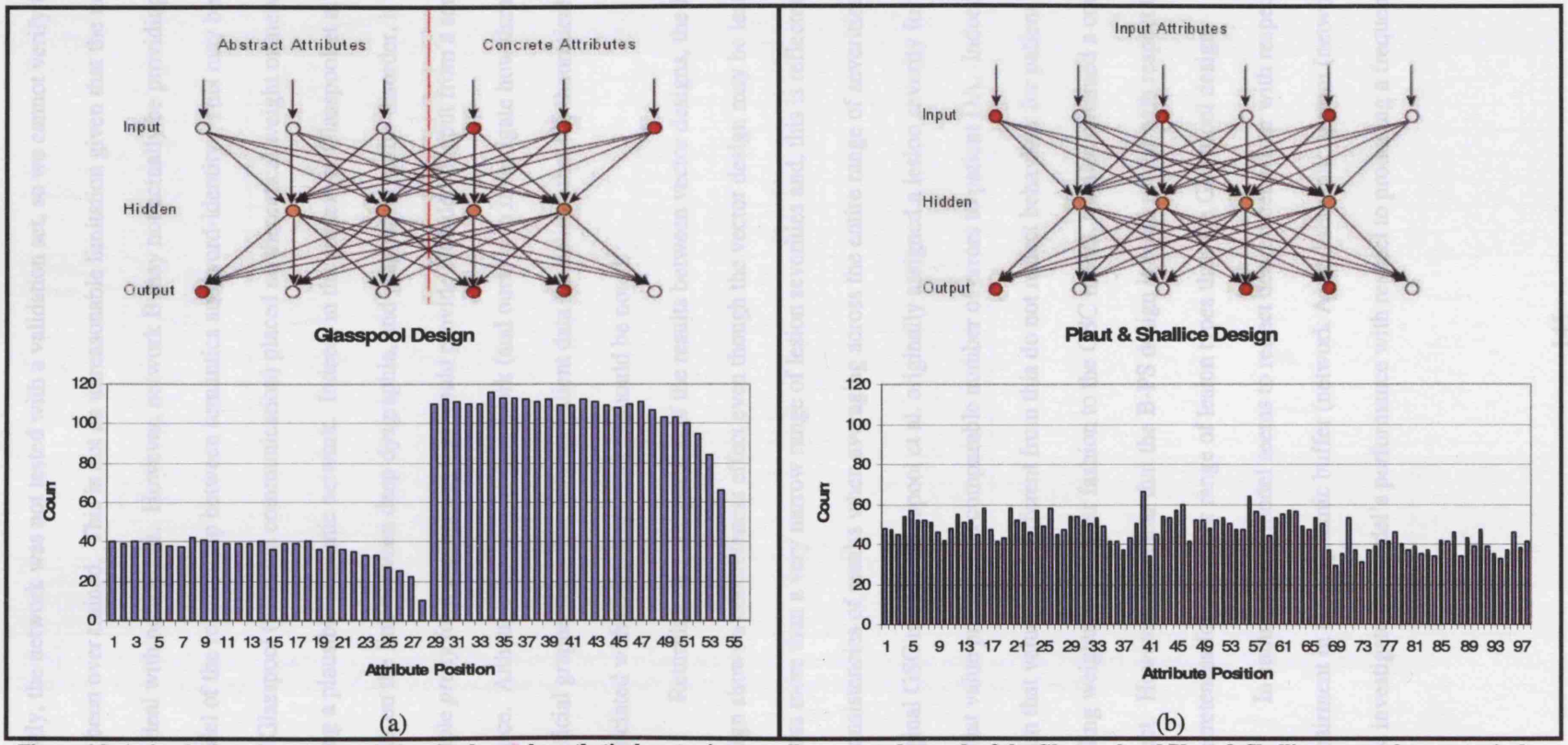



**Figure 6.3: A simple three layer network showing how a hypothetical vector is generally presented to each of the Glasspool and Plaut & Shallice approaches respectively. Red circles indicate ON values, white circles indicate OFF values, and orange circles indicate an intermediate level of activity. The histograms beneath each network show the sum of ON values (count) for each attribute position produced by the Glasspool, and Plaut & Shallice approaches respectively for each 400 word corpus.**

Firstly, the network was not tested with a validation set, so we cannot verify that it has not been over trained. This is not an unreasonable limitation given that the network does not deal with non-words. However, network B may not actually be providing a general model of the relationship between semantics and word-identity. This may be a concern, but Glasspool (personal communication) placed no theoretical weight on network B being a plausible semantic network. Indeed, in the context of Glasspool et al's original work on the link between deep dysgraphia, and graphemic buffer disorder, it was a simple *proxy* for a mechanism that would provide disordered input from a semantic source. Although the crux of their work (and ours) is to investigate how damage to an artificial graphemic buffer reflects patient data we believe that the theoretical issues associated with training network B should be noted.

Returning to a comparison of the results between vector designs, the B-DG design shows a concreteness effect even though the vector design may be less robust across more than a very narrow range of lesion severities and, this is reflected by inconsistencies of results when averaging across the entire range of severities. In the original GSC model, Glasspool et al. originally assigned a lesion severity for network B as that value producing a comparable number of errors to patient DA. Indeed, one could claim that values very different from this do not reflect behaviour for patient DA. By scaling weights in a similar fashion to the GSC model, we also obtained a concreteness effect. However, it is clear that the B-PS design is more robust with respect to concreteness for a broader range of lesion types than the Glasspool design.

In summary, the model seems to reflect desired behaviour with respect to loci of impairment in the graphemic buffer (network A) and semantic system (network B). We now investigate the model's performance with respect to producing a frequency effect.

| Lesion Location | Lesion Type | A-MW | | A-IE | | A-POS | | B-DG | | B-PS | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles |
| Random | Ablate | n.s. -0.4 | -0.4, -1.6, 1.4 | n.s. 0.1 | 0.4, 1.0, 0.3 | n.s. 0.0 | 0.4, 0.0, 1.5 | ** **-8.0** | -9.1, -12.7, -6.9 | ** **-9.3** | -10.3, -11.0, -8.6 |
| | Noise | * **-0.5** | -1.5, -2.1, 0.2 | n.s. 0.5 | -0.6, -2.7, 1.9 | n.s. 0.1 | 1.9, 1.3, -0.3 | ** **-11.6** | -15.8, -17.9, -10.2 | ** **-12.5** | -15.2, -17.7, -11.8 |
| | Constrain | ** **-3.4** | -4.9, -4.7, -3.6 | n.s. -0.5 | -0.1, -1.8, -2.3 | n.s. -0.3 | -0.7, 1.5, -0.2 | ** **-9.6** | -10.5, -13.1, -9.1 | ** **-12.7** | -15.8, -16.2, -14.0 |
| | Scale | ** **-3.8** | -7.3, -5.3, -2.2 | * **-1.3** | -2.0, -3.2, -2.2 | * **-1.1** | -1.7, -3.0, -2.5 | ** **-16.1** | -19.5, -22.0, -18.8 | ** **-12.5** | -18.7, -18.7, -18.7 |
| Input to Hidden | Ablate | * **-0.8** | -0.5, -0.6, 0.6 | n.s. 0.1 | 0.9, 0.2, 0.3 | n.s. -0.1 | -2.5, 1.5, -1.1 | ** **-9.7** | -9.2, -12.7, -10.3 | ** **-9.9** | -12.5, -12.6, -8.8 |
| | Noise | n.s. 0.0 | 0.5, -0.9, 1.1 | * **0.7** | -0.4, 2.8, 0.8 | n.s. 0.5 | 2.0, 0.2, -0.6 | ** **-8.5** | -9.9, -12.1, -10.1 | ** **-8.7** | -10.0, -14.1, -8.2 |
| | Constrain | ** **-2.5** | -4.9, -2.7, -2.6 | n.s. -0.5 | 0.2, -1.3, 0.9 | ** **-1.7** | -1.9, -2.5, -1.1 | ** **-8.9** | -10.4, -10.9, -8.0 | ** **-11.4** | -14.3, -14.4, -11.3 |
| | Scale | * **-3.1** | -8.5, -0.7, -2.5 | ** **-1.2** | -1.8, -1.7, -1.0 | ** **-1.5** | -0.2, -0.7, -1.2 | ** **-13.0** | -15.3, -21.7, -12.0 | ** **-11.4** | -15.3, -13.3, -13.3 |
| Context to Hidden | Ablate | n.s. -0.7 | 0.9, 1.6, -1.9 | n.s. 0.1 | -0.9, 0.6, 0.2 | n.s. -0.1 | -0.9, 0.9, 0.1 | N/A | | | |
| | Noise | n.s. -0.5 | -0.4, -0.8, -1.2 | n.s. -0.1 | 0.4, 0.3, 0.4 | n.s. 0.4 | 2.2, -2.2, -0.4 | | | | |
| | Constrain | ** **-1.2** | -2.8, -0.4, -0.9 | * **1.4** | 0.1, 3.5, 2.6 | n.s. -0.3 | -1.9, 0.1, 1.7 | | | | |
| | Scale | n.s. -0.5 | -2.7, 1.0, 0.8 | * **2.1** | -0.5, 3.8, 4.7 | * **-1.5** | 1.2, -5.0, -1.7 | | | | |
| Word Identity to Hidden | Ablate | n.s. -0.4 | -0.6, -0.7, -1.7 | n.s. 0.1 | 1.9, 0.1, 0.8 | n.s. 0.0 | -0.8, 1.4, -0.5 | | | | |
| | Noise | n.s. -0.1 | 1.3, -1.4, -1.1 | n.s. 0.1 | -0.5, -0.9, 1.7 | n.s. -0.3 | 0.3, -1.8, 0.5 | | | | |
| | Constrain | ** **-3.2** | -3.8, -5.8, -2.4 | ** **-1.5** | -1.0, -1.8, -1.3 | ** **-3.7** | -4.8, -5.2, -3.7 | | | | |
| | Scale | ** **-2.6** | -3.2, -5.5, -1.5 | ** **-1.3** | -1.8, -1.3, -1.0 | ** **-4.2** | -2.8, -6.0, -4.0 | | | | |
| Hidden to Output | Ablate | n.s. -0.2 | -0.4, 0.6, -0.5 | n.s. -0.5 | -0.3, 1.3, -1.4 | n.s. 0.1 | -0.7, 0.0, -0.6 | ** **-11.5** | -10.6, -14.1, -9.5 | ** **-10.4** | -10.8, -13.4, -10.4 |
| | Noise | n.s. -0.8 | -4.0, -0.1, 0.0 | n.s. 0.0 | -0.1, -0.3, 1.8 | n.s. -0.4 | -1.5, -1.5, -0.5 | ** **-14.1** | -15.7, -19.4, -12.6 | ** **-12.2** | -18.8, -17.0, -13.8 |
| | Constrain | * **-0.8** | -2.6, -0.3, -1.5 | n.s. -0.4 | 0.0, -3.6, 0.3 | n.s. 0.5 | 0.4, 0.0, 2.0 | ** **-9.7** | -18.5, -19.2, -10.2 | ** **-10.2** | -20.6, -20.1, -10.2 |
| | Scale | ** **-1.6** | -0.8, -1.2, -4.2 | ** **-1.7** | -2.8, -2.0, -2.0 | ** **1.5** | 1.5, 1.0, 2.2 | ** **-11.6** | -18.8, -23.2, -8.5 | ** **-15.1** | -25.0, -24.3, -16.3 |
| Output | Noise | * **-0.7** | -1.2, -1.1, -0.9 | n.s. 0.0 | 0.1, 1.0, -0.1 | n.s. 0.2 | 1.0, -0.7, -0.4 | N/A | | | |
| **Average magnitude** | | **-1.3** | | **-0.2** | | **-0.6** | | **-11.0** | | **-11.4** | |
| **Significant and m≥ ±2.5** | | **6/21** | | **0/21** | | **2/21** | | **12/12** | | **12/12** | |

**Table 6.7: Low and high frequency error comparisons across lesion types, locations, and severities for total errors. Positive magnitudes indicate more high frequency errors and negative magnitudes indicate more low frequency errors. See Table 6.6 for a description of each column.**

### 6.4.2 Frequency

Pure GBD patients should show no effect of word frequency on error rates, with high frequency words being as susceptible to error as low frequency words are (e.g. LB - Caramazza et al, 1987, SE - Posteraro et al, 1988, ML - Hillis & Caramazza, 1989, AS - Jonsdottir et al, 1996, and BWN - Tainturier & Rapp, 2004). Patients with an apparent deficit in the semantic system are expected to show more errors on low frequency than high frequency words (e.g. VS - Nolan & Caramazza, 1983, TH & PB – Schiller et al, 2001, FM – Tainturier & Caramazza, 1996, and DA - Cipolotti et al, 2004). Table 6.7 compares low and high frequency error rates in the model. Our first general observation in comparing the number of significant substantial occurrences for network A (8/63) and network B (24/24) is that they behave very differently with respect to frequency ($\chi^2(1) = 56.964$, $p < 0.001$). Network B shows a clear frequency effect whereas network A shows for the most part almost no frequency effect.

As with tests for concreteness, the IE (A-IE) mechanism in network A shows a good fit to the patient data with no noticeable difference in error rates between low and high frequency words. The moving window (A-MW) mechanism still shows a tendency to produce the effect (far less however than shown with network B), and unlike tests for concreteness, the positional context seemed more prone to error. In particular, this propensity for error still appears to arise with the constraint and scale lesion types. Of the 400 words in the corpus, 100 are high frequency and concrete, 100 are low frequency and concrete, 100 are high frequency and abstract, and 100 are low frequency and abstract. Despite the apparent problems with the Glasspool design in explaining concreteness, both semantic designs produced consistently more errors for low frequency words. Low frequency words were trained with a probability of 0.30 whereas

high frequency words were always trained. The extra -training associated with half the corpus may have provided more robustness to high frequency words despite the fact that 100 of those 200 words were abstract.

As described earlier, lesion types that reduce overall contributions to hidden and output nodes (i.e. constraint and scale) tend to produce the most substantial values. This seems to be the case across both network A and network B. Where ablation and noise may alter the overall weight contributions to a node either up or down, constraint and scale each reduce the overall contribution to a node, and thus its ability to produce high activation values. The fact that these are accompanied by significant (if not substantial) differences in error rates also suggests that this effect is consistent across lesion severity.

### 6.4.3 Spelling related Analyses

We have so far investigated the effect that lesioning has on the model's ability to reflect patient data concerning concreteness and frequency. According to traditional spelling models, neither of these symptoms should be caused by damage solely within the graphemic buffer. One property of dysgraphia, which is deemed to be situated in the graphemic buffer, is that of within-class substitutions and transpositions. We now examine the effect of a consonant/vowel status associated with each letter. As this relates purely to orthography, all tests will be applied solely to network A.

#### 6.4.3.1 An Analysis of Consonant and Vowel Behaviour

Several authors have described behaviour suggesting that letter identity can be lost while CV status is preserved (e.g. Miceli, Silveri, & Caramazza, 1985, Caramazza & Miceli, 1990; McCloskey et al, 1994, Cubelli, 1991, Miceli et al 1995, Jónsdóttir et al., 1996;Caramazza, Chialant, Capasso and Miceli, 2000, Miceli, Capasso, Benvegnù & Caramazza, 2004). In LB (Caramazza & Miceli, 1990) for example, errors like

DECINE (tens)→CECINE were common, but errors like SCUOLA (school)→SCLOLA were very rare and substitutions made by CW (Cubelli, 1991) involved vowels for vowels and consonants for consonants 99% of the time. JH (Kay and Hanley, 1994) showed similar results for single errors. In cases involving substitution of consonants, 94% of the time they were replaced by another consonant and in cases involving vowels 88.5% were replaced by other vowels. In single letter substitutions by HE (McCloskey et al, 1994), 95% of these involved substitution of a consonant for a consonant, or a vowel for a vowel. Patient AS (Jonsdottir et al, 1996) substituted consonants for consonants 92% of the time, and vowels for vowels 78% of the time. A less convincing example is provided by DS (Chialant et al, 2002) whose vowel for vowel substitutions was only 39%. Error behaviour with respect to vowels or consonants is also not consistent. For example, patients AS2[19] and IFA (Caramazza et al, 2000) both showed contrasting, selective difficulties in producing vowels and consonants, with AS2 producing nearly three times as many errors on vowels as consonants and IFA producing nearly five times as many errors on consonants as vowels. It seems therefore that any model proposing to produce plausible behaviour should behave accordingly. Although the GSC model includes CV nodes as described in Chapter 5, we expand on Glasspool's (1998) analysis so as to provide a further foundation for explorations into orthographic complexity further in the chapter.

### 6.4.3.2 Chance Behaviour

We first investigate the relationship between consonants and vowels in our corpus before interpreting their behaviour in the model. By calculating letter frequencies, we can determine the probabilities of a letter substituting with any other letter. Equation 6.2

[19] AS2 is cited in the literature by Caramazza et al (2000) as patient AS. However, we distinguish between their AS, a 41 year old woman, and AS (Jonsdottir et al, 1996), a 68 year old man we refer to more often.

and Equation 6.3 respectively describe the probabilities of a specific consonant or vowel appearing in our corpus.

$$P(C_i) = \frac{\sum_i C_i}{\sum_i C_i + \sum_j V_j} \text{ and} \qquad \textbf{Equation 6.2}$$

$$P(V_j) = \frac{\sum_j V_j}{\sum_i C_i + \sum_j V_j} \qquad \textbf{Equation 6.3}$$

where $C_i$ and $V_j$ refer to specific instances of consonants and vowels, and $i$ and $j$ are indices for the set of consonant and vowels respectively. Therefore, the probability of a letter being either a consonant or vowel respectively is:

$$P(C) = \sum_i P(C_i) \text{ and} \qquad \textbf{Equation 6.4}$$

$$P(V) = \sum_j P(V_j) \qquad \textbf{Equation 6.5}$$

The probability of a specific consonant substituting with a different consonant is:

$$P(C_iC) = P(C_i)(P(C) - P(C_i)) \qquad \textbf{Equation 6.6}$$

and the probability of a specific vowel substituting with a different vowel.

$$P(V_jV) = P(V_j)(P(V) - P(V_j)) \qquad \textbf{Equation 6.7}$$

The probabilities of a specific consonant substituting with a vowel, or a vowel substituting with a consonant respectively are

$$P(C_iV) = P(C_i)P(V) \qquad \textbf{Equation 6.8}$$

and

$$P(V_jC) = P(V_j)P(C) \qquad \textbf{Equation 6.9}$$

When applied to the 400 words in our corpus, Table 6.8 and Table 6.9 summarise the chance probabilities outlined in Equation 6.2 to Equation 6.9 for vowels and consonants respectively. The column labelled *Correct* is the chance of a letter substituting with

another of the same value, and provided as a checksum to ensure correct summations for the values of $\sum P(C_i)$ and $\sum P(V_j)$.

| Letter | N | $P(V_i)$ | $P(V_iV)$ | $P(V_iC)$ | Correct |
|---|---|---|---|---|---|
| A | 185 | 0.08409 | 0.02446 | 0.05256 | 0.00707 |
| E | 269 | 0.12227 | 0.03090 | 0.07642 | 0.01495 |
| I | 142 | 0.06455 | 0.02004 | 0.04034 | 0.00417 |
| O | 148 | 0.06727 | 0.02070 | 0.04205 | 0.00453 |
| U | 81 | 0.03682 | 0.01245 | 0.02301 | 0.00136 |
| **Sum** | **825** | **0.37499** | **0.10855** | **0.23438** | **0.03208** |

**Table 6.8: Probability table relating to vowels**

| Letter | N | $P(C_i)$ | $P(C_iC)$ | $P(C_iV)$ | Correct |
|---|---|---|---|---|---|
| B | 42 | 0.01909 | 0.01157 | 0.00716 | 0.00036 |
| C | 106 | 0.04818 | 0.02779 | 0.01807 | 0.00232 |
| D | 68 | 0.03091 | 0.01836 | 0.01159 | 0.00096 |
| F | 33 | 0.01500 | 0.00915 | 0.00562 | 0.00022 |
| G | 51 | 0.02318 | 0.01395 | 0.00869 | 0.00054 |
| H | 72 | 0.03273 | 0.01938 | 0.01227 | 0.00107 |
| J | 6 | 0.00273 | 0.00170 | 0.00102 | 0.00001 |
| K | 27 | 0.01227 | 0.00752 | 0.00460 | 0.00015 |
| L | 100 | 0.04545 | 0.02634 | 0.01705 | 0.00207 |
| M | 60 | 0.02727 | 0.01630 | 0.01023 | 0.00074 |
| N | 137 | 0.06227 | 0.03504 | 0.02335 | 0.00388 |
| P | 72 | 0.03273 | 0.01938 | 0.01227 | 0.00107 |
| Q | 8 | 0.00364 | 0.00226 | 0.00136 | 0.00001 |
| R | 200 | 0.09091 | 0.04855 | 0.03409 | 0.00826 |
| S | 116 | 0.05273 | 0.03017 | 0.01977 | 0.00278 |
| T | 155 | 0.07045 | 0.03907 | 0.02642 | 0.00496 |
| V | 32 | 0.01455 | 0.00888 | 0.00545 | 0.00021 |
| W | 30 | 0.01364 | 0.00834 | 0.00511 | 0.00019 |
| X | 4 | 0.00182 | 0.00113 | 0.00068 | 0.00000 |
| Y | 52 | 0.02364 | 0.01421 | 0.00886 | 0.00056 |
| Z | 4 | 0.00182 | 0.00113 | 0.00068 | 0.00000 |
| **Sum** | **1375** | **0.62501** | **0.36022** | **0.23434** | **0.03036** |

**Table 6.9: Probability table relating to consonants**

Using values in Table 6.8 and Table 6.9, $P(C) = 0.625$ and $P(V) = 0.375$. Comparing actual versus chance behaviour shows some interesting results. Table 6.10, for example details the behaviour of our model trained without CV nodes. The results are comparable to chance expectations (Equation 6.2 to Equation 6.9) as we would not expect an overt influence of consonant or vowel status in errors associated with substitutions and transpositions.

| Lesion Loc. | Lesion Type | Substitutions | | | | | | | Transpositions | | | | | Inserts | | | Del. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | % | WW | Vow | CC | VV | CV | VC | % | WW | CC | CV [20] | VV | % | WW | Vow | % | Vow |
| Random | Ablate | 46.7 | 57 | 35 | 44 | 10 | 21 | 25 | 30.6 | 69 | 21 | 71 | 8 | 1.9 | 68 | 50 | 20.8 | 31 |
| | Noise | 47.8 | 61 | 45 | 40 | 12 | 16 | 32 | 30.5 | 78 | 19 | 64 | 17 | 2.4 | 65 | 38 | 19.3 | 35 |
| | Constrain | 36.3 | 80 | 50 | 40 | 10 | 10 | 40 | 19.3 | 81 | 24 | 70 | 6 | 0.7 | 57 | 41 | 43.8 | 31 |
| | Scale | 37.5 | 78 | 53 | 42 | 5 | 5 | 48 | 16.5 | 79 | 33 | 63 | 4 | 1.3 | 26 | 20 | 44.8 | 37 |
| Input to Hidden | Ablate | 47.8 | 59 | 36 | 42 | 12 | 22 | 24 | 31.8 | 72 | 22 | 70 | 8 | 1.6 | 63 | 42 | 18.8 | 34 |
| | Noise | 47.2 | 63 | 42 | 42 | 10 | 17 | 31 | 32.5 | 77 | 19 | 74 | 7 | 1.4 | 67 | 42 | 19.0 | 35 |
| | Constrain | 43.6 | 76 | 48 | 42 | 9 | 10 | 39 | 25.7 | 82 | 21 | 71 | 8 | 1.3 | 50 | 37 | 29.4 | 32 |
| | Scale | 41.9 | 79 | 44 | 47 | 8 | 9 | 36 | 24.4 | 81 | 31 | 65 | 4 | 1.7 | 54 | 42 | 32.1 | 27 |
| Context to Hidden | Ablate | 48.6 | 59 | 33 | 45 | 12 | 22 | 21 | 33.3 | 71 | 24 | 69 | 7 | 1.8 | 70 | 40 | 16.3 | 34 |
| | Noise | 45.0 | 63 | 46 | 36 | 14 | 18 | 32 | 32.5 | 76 | 18 | 74 | 8 | 1.7 | 61 | 34 | 20.8 | 38 |
| | Constrain | 47.7 | 78 | 39 | 46 | 10 | 15 | 29 | 32.0 | 84 | 30 | 61 | 9 | 1.5 | 69 | 46 | 18.8 | 28 |
| | Scale | 47.1 | 83 | 33 | 52 | 9 | 15 | 24 | 33.3 | 84 | 33 | 62 | 5 | 2.8 | 55 | 42 | 16.9 | 26 |
| Word Identity to Hidden | Ablate | 48.3 | 55 | 39 | 42 | 13 | 19 | 26 | 29.4 | 66 | 22 | 71 | 8 | 1.6 | 71 | 45 | 20.8 | 32 |
| | Noise | 46.7 | 63 | 44 | 40 | 10 | 17 | 33 | 31.1 | 73 | 24 | 67 | 9 | 1.9 | 74 | 37 | 20.4 | 35 |
| | Constrain | 38.2 | 61 | 58 | 37 | 8 | 5 | 50 | 21.1 | 72 | 25 | 68 | 7 | 1.8 | 72 | 50 | 39.0 | 28 |
| | Scale | 38.2 | 61 | 60 | 36 | 8 | 4 | 52 | 20.2 | 72 | 27 | 67 | 6 | 1.4 | 65 | 56 | 40.1 | 28 |
| Hidden to Output | Ablate | 47.5 | 57 | 36 | 45 | 10 | 19 | 26 | 31.2 | 70 | 23 | 70 | 7 | 2.0 | 68 | 44 | 19.3 | 31 |
| | Noise | 49.2 | 60 | 41 | 45 | 8 | 14 | 33 | 31.7 | 77 | 22 | 73 | 5 | 2.2 | 60 | 40 | 16.9 | 32 |
| | Constrain | 31.7 | 68 | 54 | 33 | 10 | 14 | 43 | 10.5 | 86 | 16 | 74 | 10 | 1.1 | 63 | 22 | 56.6 | 39 |
| | Scale | 0.5 | 100 | 63 | 6 | 28 | 32 | 34 | 0.0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0 | 99.5 | 32 |
| Output | Noise | 42.9 | 63 | 38 | 42 | 10 | 20 | 28 | 35.3 | 78 | 21 | 70 | 9 | 1.8 | 59 | 39 | 20.0 | 32 |
| **Chance value** | | **N/A** | **N/A** | **37.5** | **36.0** | **10.8** | **23.4** | **23.4** | **N/A** | **N/A** | **36.0** | **46.8** | **10.8** | **N/A** | **N/A** | **37.5** | **N/A** | **37.5** |
| Mean (actual values) | | 41.9 | 67.8 | **44.6** | **40.2** | **10.8** | **15.4** | **33.6** | 26.3 | 72.8 | **22.6** | **65.4** | **7.2** | 1.6 | 58.9 | **38.4** | 30.1 | **32.2** |
| Median | | 46 | 63 | 44 | 42 | 10 | 16 | 32 | 31 | 77 | 22 | 70 | 7 | 1.7 | 63 | 41 | 21 | 32 |
| St Deviation | | 10.6 | 11.5 | 8.9 | 8.9 | 4.4 | 6.7 | 8.9 | 8.9 | 17.5 | 7.0 | 15.5 | 3.2 | 0.6 | 17.0 | 12.0 | 19.6 | 3.6 |

**Table 6.10 : Network A trained using a moving window context and no CV nodes. Values describe proportion of letters in error and are based on total errors. For definitions, see the text on the following page.**

[20] For Transpositions, we do not differentiate between consonants transposed with vowels and vice-versa as we cannot determine which letter is responsible for the transposition.

Columns titled **%** indicate the proportion of error type. In the first row for example, we see that random ablation produces 46.7% substitutions, 30.6% transpositions, 1.9% of inserts and 20.8% of deletes. The column **WW** (within-word) shows the proportion of substitutions, transpositions, and inserts involving a letter also present in the original word; all deletes are naturally within-word. The **Vow** sub-columns define how many vowels are inserted, deleted, or result from a substitution, with the complement thus representing the proportion of consonants in error. In the **Substitutions** column, **CC**, **VV**, **CV**, and **VC** represent the percentage of Consonants substituted by a Consonant, Vowels substituted by a Vowel, Consonants substituted by a Vowel and Vowels substituted by a Consonant respectively. In the **Transpositions** column, **CC**, **CV**, and **VV** represent the percentage of Consonants transposing with a Consonant, Consonants transposing with a Vowel, and Vowels transposing with another Vowel respectively. Unlike substitutions, it is not possible to determine which transposed letter position is accountable for the transposition. Hence, we do not discriminate between a consonant transposing with a vowel and a vowel transposing with a consonant and cannot provide a **Vow** column.

The first observation regarding Table 6.10 is that the similar lesion approaches tend to produce comparable behaviour across lesion location, ablation being similar to noise and constraining weights being similar to scaling. This can be seen in terms of their relative error distributions and in some instances CV behaviour. Most lesion locations seem to behave similarly with respect to how each individual lesion type affects error distribution, and CV error distribution. Instead of performing exhaustive tests on each lesion location and type, we have chosen to use the 'Word Identity to Hidden' lesion location as indicative of the behaviour one might expect. The proportion of substitutions, transpositions, and deletes when lesioning from Word Identity to

Hidden nodes, shows very similar values for Ablate and Noise lesion types and for Constrain and Scale types respectively. Table 6.11 for example shows a strong statistical relationship between error distributions[21]

| Lesion Type | Ablate | Noise | Constrain | Scale |
|---|---|---|---|---|
| **Ablate** | - | ** (ρ=0.998) | n.s. (ρ=0.759) | n.s. (ρ=0.737) |
| **Noise** | | - | n.s. (ρ=0.741) | n.s. (ρ=0.717) |
| **Constrain** | | | - | ** (ρ=0.919) |
| **Scale** | | | | - |

**Table 6.11: Correlation matrix of error percentage by lesion type from *Word Identity to Hidden*.**

It seems intuitive that ablation and noise would behave similarly, as would constraint and scaling. The two former techniques slightly alter the overall weight contribution to each node's activation function (up or down), whereas the two latter reduce the overall weight contributions to a node's activation function. The probabilities outlined in Equation 6.2 to Equation 6.9 are chance in nature as they do not account for the effect of the model to promote letters to substitute, transpose, or be inserted if already present in the word. Indeed, the model almost *primes* a letter to be ready to be produced the closer it is to its expected position. Conversely, letters unrelated to the current word are free to retain any non-participatory activation, which is usually manifested by their activations being comparatively lower than those of letters within the current word. Although we have not calculated chance values for within word substitutions, actual values in Table 6.10 range from 59% to 73%. Patients have also been shown to produce a qualitatively similar propensity to substitute with letters already present in the word. Patient AS's (like LB's) substitutions tended to involve letters also within the word such as SINCERE → CINCERE, yet there are exceptions to this behaviour. For patient JH (Kay & Hanley, 1994) the majority of substituting letters did not occur elsewhere in the word yet bore a distinct visual similarity to the target. Patient VS (Nolan & Caramazza, 1983) also made

[21] Each lesion type was correlated against each other comparing the proportion of insert, substitutions, transpositions, and deletes. For example, Ablate (48.6, 33.3, 1.8, 16.3) → (Noise: 45.0, 32.5, 1.7, 20.8).

substitutions based on visual similarities (e.g. 'n' for 'r' and 'r' for n'). Errors involving substitutions with visual similarity suggest a different functional locus of impairment than that shown by AS and LB, possibly later than the graphemic buffer.

The number of substitutions related to CV class seems close to chance (CC → 40.2%/36.0%, VV → 10.8%/10.8%, CV → 15.4%/23.4%, VC → 33.6%/23.4%). However, transpositions show many more out of class interactions (CV → 65.4%), and may be explained by examining the actual corpus structure. Most transpositions in English are likely to be with neighbouring positions. In our corpus, there are 1800 possible neighbouring transpositions. Of these 526 (29%) would be within class, and the remaining 1274 (71%) out of class. Table 6.10 gives a similar result (22.6+7.2 = 30% and 65.4%). In considering transpositions between letters two positions apart, the probabilities change. Of 1400 possible transpositions, 41 were ignored as they would involve substitution with another letter of the same value. Of the remaining 1359, 666 (47.6%) would be within class, and 693 (49.5%) out of class, which is very close to chance (46.8% and 46.8%). Table 6.8 and Table 6.9 relate to completely random letter combinations, whereas our network is trained on a corpus containing orthotactically valid English words. As English is a structured language, we assume that the network has learned to generalise English-*like* CV structures, for example a dominance of VCV structures at the end of words such as EMPIRE (VCCVCV), or JUSTICE (CVCCVC) in our corpus.

An alternative explanation for the comparatively fewer within-class transpositions than Table 6.8 and Table 6.9 suggest, may be due to our definition of a transposition. Many analyses of transpositions in the literature categorise them as such, where it is obvious that two letters have exchanged places, for example ALZARE → AZLARE (Caramazza & Miceli, 1990). We also count complex transpositions

involving a substitution for example ALTARE → ARLARE (Caramazza & Miceli, 1990). Here, the L and T transpose, and the T substitutes with a within-word consonant. We argue that this is still a valid transposition albeit complex, rather than a delete and an addition. Our reasons are firstly that substitutions are much more prevalent in patient data than deletes and inserts, and secondly that a single substitution is a simpler operation than a coordinated delete and insert involving the same letter position. In terms of actual outputs, a coordinated delete and insert of the correct letters is indistinguishable from a transposition.

### 6.4.3.3 The effect of training with CV nodes.

We now examine the effect of training the model with CV nodes, and the results of lesioning the model can be seen in Table 6.12. Note firstly, the difference in error distributions. Average substitutions have increased (41.9%→54.7%), transpositions decreased (26.3%→12.3%), inserts decreased (1.6%→0.2%), and deletes increased (30.1%→34.5%). Our corpus has 526 instances of letters next to a letter of the same CV class, and 1275 instances of letters next to a letter of the other CV class.

Since most neighbouring letters are of a different class, we argue that a reduction in transpositions shows CV class having an inhibitory effect on out of class transpositions. Note also the increase of within-class substitutions from 51% (CC of 40.2 + VV of 10.8) in Table 6.10 to 81.3% (CC of 55.8 + VV of 25.5) in Table 6.12, and within-class transpositions from 29.8% (CC of 22.6 + VV of 7.2) to 70.4% (CC of 48.4 + VV of 22.0).

| Lesion Location | Lesion Type | Substitutions | | | | | | | Transpositions | | | | | Inserts | | | Del. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | % | WW | Vow | CC | VV | CV | VC | % | WW | CC | CV | VV | % | WW | Vow | % | Vow |
| Random | Ablate | 63.6 | 52 | 28 | 59 | 26 | 13 | 2 | 17.2 | 68 | 51 | 24 | 25 | 0.4 | 72 | 25 | 18.9 | 22 |
| | Noise | 66.5 | 56 | 27 | 60 | 26 | 13 | 1 | 17.7 | 71 | 52 | 24 | 24 | 0.2 | 64 | 17 | 15.7 | 22 |
| | Constrain. | 36.0 | 77 | 25 | 57 | 24 | 18 | 1 | 5.7 | 83 | 48 | 27 | 25 | 0.1 | 98 | 7 | 58.1 | 29 |
| | Scale | 40.6 | 84 | 27 | 60 | 26 | 13 | 1 | 2.6 | 80 | 53 | 34 | 13 | 0.1 | 100 | 0 | 56.7 | 27 |
| Input to Hidden | Ablate | 61.8 | 54 | 25 | 59 | 23 | 16 | 2 | 18.8 | 64 | 45 | 32 | 23 | 0.3 | 74 | 18 | 19.2 | 25 |
| | Noise | 66.3 | 60 | 29 | 57 | 28 | 14 | 1 | 16.7 | 74 | 53 | 19 | 28 | 0.1 | 73 | 13 | 16.9 | 29 |
| | Constrain. | 55.5 | 75 | 24 | 57 | 24 | 17 | 1 | 10.7 | 82 | 55 | 24 | 21 | 0.2 | 96 | 15 | 33.7 | 27 |
| | Scale | 54.3 | 78 | 24 | 60 | 23 | 16 | 1 | 8.1 | 80 | 57 | 26 | 17 | 0.2 | 100 | 33 | 37.5 | 30 |
| Context to Hidden | Ablate | 59.9 | 55 | 24 | 54 | 21 | 22 | 3 | 18.4 | 64 | 41 | 37 | 22 | 0.4 | 67 | 24 | 21.4 | 30 |
| | Noise | 67.0 | 62 | 28 | 59 | 27 | 13 | 1 | 14.3 | 76 | 59 | 17 | 24 | 0.2 | 79 | 27 | 18.4 | 26 |
| | Constrain. | 63.9 | 77 | 23 | 55 | 23 | 21 | 1 | 13.8 | 82 | 55 | 26 | 19 | 0.4 | 91 | 8 | 21.9 | 36 |
| | Scale | 63.8 | 79 | 22 | 56 | 22 | 21 | 1 | 12.1 | 77 | 53 | 28 | 19 | 0.5 | 95 | 13 | 23.7 | 32 |
| Word Identity to Hidden | Ablate | 59.4 | 53 | 25 | 58 | 24 | 16 | 2 | 15.9 | 65 | 46 | 29 | 25 | 0.2 | 65 | 21 | 24.4 | 26 |
| | Noise | 65.7 | 58 | 28 | 59 | 27 | 13 | 1 | 16.4 | 65 | 50 | 23 | 27 | 0.4 | 68 | 13 | 17.5 | 24 |
| | Constrain. | 44.0 | 62 | 19 | 60 | 18 | 21 | 1 | 5.9 | 74 | 46 | 35 | 19 | 0.1 | 98 | 31 | 50.0 | 23 |
| | Scale | 42.5 | 62 | 17 | 61 | 17 | 21 | 1 | 5.5 | 72 | 45 | 37 | 18 | 0.1 | 100 | 37 | 51.9 | 21 |
| Hidden to Output | Ablate | 63.4 | 52 | 26 | 62 | 25 | 11 | 2 | 17.2 | 68 | 53 | 23 | 24 | 0.4 | 71 | 30 | 19.0 | 20 |
| | Noise | 67.2 | 56 | 29 | 59 | 28 | 12 | 1 | 17.0 | 73 | 59 | 18 | 23 | 0.2 | 56 | 19 | 15.6 | 23 |
| | Constrain. | 42.9 | 75 | 47 | 43 | 46 | 10 | 1 | 5.4 | 80 | 45 | 20 | 35 | 0.3 | 78 | 20 | 51.7 | 29 |
| | Scale | 1.1 | 100 | 34 | 23 | 25 | 43 | 9 | 0.0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0 | 98.9 | 23 |
| Output | Noise | 63.1 | 60 | 33 | 54 | 32 | 13 | 1 | 18.9 | 77 | 50 | 19 | 31 | 0.2 | 78 | 26 | 17.8 | 24 |
| Chance value | | N/A | N/A | 37.5 | 36.0 | 10.8 | 23.4 | 23.4 | N/A | N/A | 36.0 | 46.8 | 10.8 | N/A | N/A | 37.5 | N/A | 37.5 |
| Mean (actual values) | | 54.7 | 66.0 | 26.9 | 55.8 | 25.5 | 17.1 | 1.7 | 12.3 | 70.2 | 48.4 | 24.9 | 22 | 0.2 | 77.3 | 18.9 | 34.5 | 26.1 |
| Median | | 62 | 62 | 26 | 58 | 25 | 16 | 1 | 14 | 74 | 51 | 24 | 23 | 0.2 | 78 | 19 | 24 | 26 |
| St Deviation | | 15.8 | 13.1 | 6.1 | 8.5 | 5.8 | 7.0 | 1.8 | 6.0 | 17.3 | 12.1 | 8.4 | 7.0 | 0.1 | 22.7 | 10.2 | 21.6 | 4.0 |

**Table 6.12 : Network A trained using a moving window context and using CV nodes. Values describe the percentage of letters in error of that type. For a description of each column heading see descriptive text following Table 6.10.**

A letter's CV status in the model therefore seems to affect which letters are candidates for substitution and transposition. Table 6.13 shows similar correlations between lesion types to those in Table 6.11, albeit with weaker correlations between ablation and noise, suggesting that error distribution is no longer as consistent between lesion types.

| Lesion Type | Ablate | Noise | Constrain | Scale |
|---|---|---|---|---|
| **Ablate** | - | * (ρ=0.987) | n.s. (ρ=0.743) | n.s. (ρ=0.707) |
| **Noise** | | - | n.s. (ρ=0.628) | n.s. (ρ=0.586) |
| **Constrain** | | | - | ** (ρ=0.999) |
| **Scale** | | | | - |

**Table 6.13: Correlation matrix of error percentage by lesion type from Word Identity to Hidden nodes after training with CV nodes**

To clarify the overall effect of training with CV nodes, we correlated chance behaviour (in **red**) with actual behaviour (in **blue**) for values listed in Table 6.10, and Table 6.12. Correlating values in Table 6.10 (training *without* CV nodes) resulted in a rho of 0.864, which is statistically significant at the 1% level for a sample size of 10 and suggests that CV behaviour in Table 6.10 is similar to chance. However, correlating chance and actual behaviour for values listed in Table 6.12 (training *with* CV nodes) resulted in a rho of 0.40 suggesting no statistical significance, and that learning with CV nodes differs from chance. In other words, using CV nodes has altered the model to produce consonant/vowel behaviour in line with that of patients, which in itself differs from chance behaviour. Figure 6.4 summarises error types from Table 6.10 and Table 6.12, graphically demonstrating that training with CV nodes increases within-class errors and decreases out-of-class errors.

| Type | NO CV % | CV % |
|---|---|---|
| Substitute CC | 40.2 | 55.8 |
| Substitute VV | 10.8 | 25.5 |
| Substitute CV | 15.4 | 17.1 |
| Substitute VC | 33.6 | 1.6 |
| Transpose CC | 22.6 | 48.3 |
| Transpose CV | 65.4 | 24.9 |
| Transpose VV | 7.2 | 22.0 |

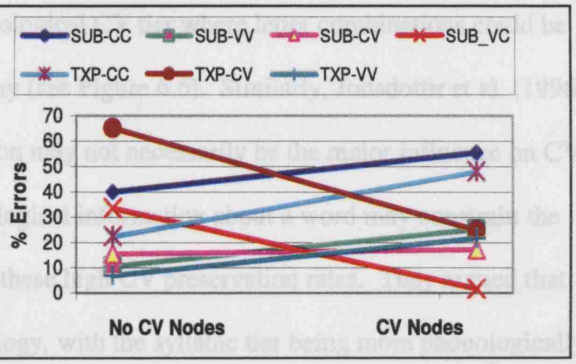



**Figure 6.4 : The effect of training with CV nodes on the relative proportion of errors**

### 6.4.4 Does orthography influence error behaviour?

It has been suggested that graphemic representations are organised in *tiers* or dimensions of information (e.g. Caramazza & Miceli, 1990, McCloskey et al, 1994, Buchwald & Rapp, 2003). In a multi-tier model, each tier identifies different aspects of a word's constituent elements. One tier for example, may refer to the word's letters, another to each letter's consonant or vowel status, another may identify whether a letter needs to be doubled, and yet another might conceivably define syllabic relationships between letters (e.g. Figure 6.5). This approach predicts that tiers may constrain the nature of errors made. As discussed in Section 6.4.3.3, a CV status promoted more within-class substitutions and transpositions, and one might expect comparable within-syllable errors were this to exist in the model. In Chapter 7, we discuss how a quantity tier predisposes double letters to be erroneous in a way consistent with a selective loss of geminate status.

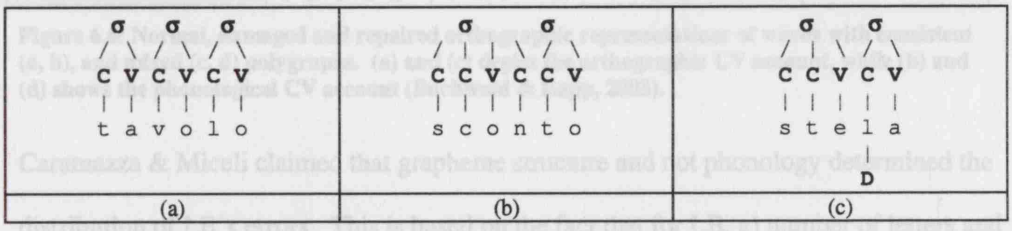



**Figure 6.5: Caramazza & Miceli's (1990) multidimensional structure. The top tier represents *graphosyllabic* boundaries, the next tier identifies a consonant/vowel status, the third tier is grapheme identity, and the last tier (showing a D) is the quantity tier for doubles.**

Buchwald & Rapp proposed a phonological CV tier where letter combinations could be phonologically related to orthography (see Figure 6.6). Similarly, Jonsdottir et al. (1996) claimed that orthographic information may not necessarily be the major influence on CV error behaviour, arguing that phonological information about a word may constrain the output in cases of damage, yielding these high CV preservation rates. They argued that LB may have relied more on phonology, with the syllabic tier being more phonologically than orthographically based, and that the regularity of Italian is a significant factor in this that would not be as pronounced in English. The key difference between Jonsdottir et al, and Caramazza & Miceli therefore seems to be that for Caramazza & Miceli syllables were segmented orthographically, whereas Jonsdottir et al assumed that this was done phonologically. Figure 6.6 indicates how orthographic and phonological repair strategies may account for CV preservation. Note in Figure 6.6 (d) how the consonant **G** is replaced by an out of word vowel contrary to a within-class strategy, yet retaining the phonological (vowel-like) nature of the syllable.

| | **Normal** | **Damaged** | | **Repaired** | |
|---|---|---|---|---|---|
| **(a) Orthographic CV Tier**<br>**Letter Identity Tier** | C C V V C<br>\| \| \| \| \|<br>C H A I N | C C V V C<br>\| \| \| \| \|<br>C H A **?** N | → | C C V V C<br>\| \| \| \| \|<br>C H A **O** N | Consistent Poly-Graphs |
| **(b) Phonological CV Tier**<br>**Letter Identity Tier** | C V C<br>/ \ / \ \|<br>C H A I N | C V C<br>/ \ / \ \|<br>C H A **?** N | → | C V C<br>/\ /\ \|<br>C H A **O** N | |
| **(c) Orthographic CV Tier**<br>**Letter Identity Tier** | C C V C C<br>\| \| \| \| \|<br>T H I G H | C C V C C<br>\| \| \| \| \|<br>T H I **?** H | → | C C V C C<br>\| \| \| \| \|<br>T H I **C** H | Mixed Poly-Graphs |
| **(d) Phonological CV Tier**<br>**Letter Identity Tier** | C V<br>/ \ / \| \<br>T H I G H | C V<br>/ \ /\|\<br>T H I **?** H | → | C V<br>/ \ /\|\<br>T H I **O** H | |

**Figure 6.6: Normal, damaged and repaired orthographic representations of words with consistent (a, b), and mixed (c, d) polygraphs. (a) and (c) depict the orthographic CV account, while (b) and (d) shows the phonological CV account (Buchwald & Rapp, 2003).**

Caramazza & Miceli claimed that grapheme structure and not phonology determined the distribution of LB's errors. This is based on the fact that for LB, a) number of letters and not number of syllables was the principle determinant of spelling performance, b) consonant substitutions were not determined by phonological similarity, c) substitution,

insertion, and transposition errors violated the sonority principle (phonotactic constraints), d) graphemic units and not phonemic units were the determinants of spelling performance, and e) spelling units such as sc →/ʃ/ and gn→/ñ/ acted like any other CC cluster with errors even violating phonotactic constraints. Caramazza & Miceli also claimed that for LB, simple CV words were less erroneous than complex CV words. We argue that the claims related to LB's performance are also consistent with the model's behaviour in the presence of damage. Without a phonological mechanism, orthography alone must be held to account for these effects. We now look specifically at how CV complexity affects propensity for error.

| Lesion Location | Lesion Type | A-MW | | A-IE | | A-POS | | B-DG | | B-PS | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles | Sig. m | Quartiles |
| Random | Ablate | ** -12.7 | -4.9, -16.0, -13.7 | ** -10.9 | -10.8, -9.4, -12.2 | ** -13.7 | -14.2, -18.2, -16.2 | ** -4.0 | -3.6, -6.2, -3.8 | ** -6.0 | -4.4, -9.9, -6.0 |
| | Noise | ** -11.3 | -14.2, -17.7, -10.1 | ** -12.1 | -16.2, -14.5, -13.3 | ** -12.6 | -11.9, -20.1, -11.3 | ** -4.5 | -3.6, -11.1, -3.9 | ** -6.3 | -5.6, -13.0, -10.0 |
| | Constrain | ** -2.1 | -0.2, 0.1, -1.4 | ** -13.7 | -19.5, -26.9, -25.5 | * -1.7 | -0.9, -1.1, -4.5 | ** -3.2 | -2.6, -2.4, -7.2 | ** -8.9 | -13.5, -16.4, -10.8 |
| | Scale | n.s. 0.8 | 4.0, 7.3, 1.1 | * -18.2 | -19.3, -35.5, -33.3 | * 1.6 | 2.5, 2.3, 2.5 | ** -8.1 | -7.9, -10.2, -15.1 | ** -9.4 | -17.8, -23.2, -23.2 |
| Input to Hidden | Ablate | ** -12.8 | -13.2, -17.8, -8.7 | ** -10.3 | -9.5, -11.5, -10.2 | ** -15.6 | -9.4, -21.2, -16.2 | ** -2.8 | -1.4, -2.4, -4.8 | ** -6.0 | -4.4, -6.4, -6.2 |
| | Noise | ** -12.6 | -14.2, -22.2, -14.3 | ** -11.5 | -10.7, -16.8, -15.7 | ** -11.1 | -10.4, -15.3, -13.7 | ** -3.0 | -3.3, -5.8, -5.0 | ** -5.9 | -7.1, -7.4, -10.3 |
| | Constrain | ** -5.6 | -10.2, -10.3, -4.4 | ** -12.5 | -12.6, -20.3, -20.7 | ** -4.4 | -4.4, -3.9, -3.5 | ** -1.5 | -0.8, -2.1, -2.9 | ** -7.3 | -6.0, -9.5, -10.9 |
| | Scale | ** -3.3 | -6.1, -3.2, -5.7 | ** -13.4 | -13.5, -24.2, -24.7 | * -2.4 | -3.0, -4.8, -1.7 | n.s. -1.7 | 2.8, -1.1, -6.8 | ** -7.3 | -13.3, -17.0, -17.0 |
| Context to Hidden | Ablate | ** -11.0 | -13.9, -12.7, -12.9 | ** -9.2 | -5.2, -9.6, -10.1 | ** -11.8 | -14.7, -13.4, -9.3 | N/A | | | |
| | Noise | ** -13.2 | -14.8, -19.5, -17.7 | ** -10.6 | -11.1, -12.7, -15.0 | ** -13.0 | -11.1, -12.1, -16.2 | | | | |
| | Constrain | ** -7.1 | -9.8, -13.2, -7.8 | ** -8.0 | -11.3, -16.2, -11.5 | ** -3.9 | -10.3, -5.1, -1.6 | | | | |
| | Scale | ** -6.0 | -14.0, -12.6, 0.0 | ** -6.2 | -5.7, -8.5, -9.6 | n.s. -2.1 | -5.1, -3.8, -2.0 | | | | |
| Word Identity to Hidden | Ablate | ** -14.7 | -15.6, -17.2, -15.1 | ** -9.7 | -10.4, -13.4, -7.9 | ** -14.9 | -8.4, -18.7, -18.0 | | | | |
| | Noise | ** -11.6 | -13.7, -17.8, -17.2 | ** -12.6 | -12.7, -19.2, -13.7 | ** -11.5 | -13.3, -18.6, -14.7 | | | | |
| | Constrain | ** -17.5 | -22.5, -26.0, -23.9 | ** -12.0 | -9.8, -16.2, -20.1 | ** -10.9 | -11.3, -15.6, -9.5 | | | | |
| | Scale | ** -18.1 | -24.9, -27.9, -26.7 | ** -12.9 | -11.0, -11.8, -19.8 | ** -10.3 | -9.3, -18.9, -13.1 | | | | |
| Hidden to Output | Ablate | ** -13.3 | -13.4, -14.0, -11.7 | ** -12.8 | -11.4, -15.0, -14.6 | ** -13.5 | -11.0, -12.1, -13.6 | ** -3.1 | -2.5, -3.9, -4.6 | ** -7.0 | -7.9, -10.6, -6.5 |
| | Noise | ** -10.7 | -10.0, -16.4, -14.6 | ** -10.8 | -11.9, -18.8, -12.8 | ** -13.0 | -18.6, -17.7, -12.4 | ** -6.0 | -7.4, -10.9, -8.2 | ** -6.1 | -10.1, -7.8, -6.3 |
| | Constrain | ** -2.8 | -0.1, -7.4, -3.8 | ** -14.5 | -15.5, -26.5, -19.6 | ** -4.0 | -4.4, -4.4, -3.6 | ** -4.9 | -5.8, -11.2, -9.8 | ** -8.2 | -13.8, -16.9, -13.8 |
| | Scale | ** 4.5 | 5.3, 5.9, 4.4 | ** -23.4 | -33.0, -33.7, -34.9 | * 3.0 | 0.4, 9.6, 1.5 | ** -4.3 | -3.5, -5.8, -7.6 | ** -12.6 | -16.4, -21.0, -14.9 |
| Output | Noise | ** -11.7 | -11.9, -14.6, -11.5 | ** -8.9 | -10.7, -14.0, -8.3 | ** -14.1 | -14.3, -18.5, -14.0 | N/A | | | |
| **Average magnitude** | | **-9.2** | | **-12.1** | | **-8.6** | | **-3.9** | | **-7.6** | |
| **Significant and m≥ ±2.5** | | **19/21** | | **21/21** | | **17/21** | | **10/12** | | **12/12** | |

**Table 6.14: Simple and complex CV error comparisons across lesion types, locations, and severities. Positive magnitudes indicate more errors associated with simple CV words and negative magnitudes indicate more errors associated with complex CV words. See Table 6.6 for a description of each column.**

As with our comparisons of concreteness and frequency, Table 6.14 compares the error rates associated with simple and complex CV words. Our first observation comparing the number of significant substantial occurrences from network A (57/63) with network B (22/24) is that there was no statistical difference (Yates' $\chi^2$ (1) = 0.059, p > 0.808). In addition, all but four magnitudes across all 87 tests were negative suggesting that the effect is consistent (if not always substantial) across both networks.

Network A shows no significant difference between the number of substantial significant occurrences (19/21, 21/21, 17/21) across all serial context types (Fisher's Exact = 0.15). It is interesting to note that in cases where the differences were not substantial, these are typically associated with constraint and scale lesions. Comparing substantial significant occurrences (10/12, 12/12) across both semantic designs in network B also shows no significant difference in producing the CV-complexity effect (Yates' $\chi^2$ (1) = 0.515, p > 0.46), and like network A, instances where the difference was not substantial enough involved constraint and scale lesions.

Our overall conclusion regarding the effect of scale and lesion types with respect to CV complexity must be to start with the assumption that producing more errors on complex than simple CV words is *normal* behaviour for the model and that simple CV words are in fact more robust than complex CV words. Therefore, while ablation and noise tend to disrupt the model's ability to function perfectly, it copes well, producing errors in line with a CV-complexity effect. Scale and constraint lesion types also deliver significant effects but these are not generally as substantial as with ablation and noise. The process of indirectly reducing the activation of hidden and output nodes may therefore affect the model's ability to robustly produce *normal* behaviour. This may also explain the two positive significant magnitudes occurred as an effect of scaling.

It is debatable however, whether damage to the lexical-semantic area can be held accountable for orthographic errors. After all, word identity held in the orthographic lexicon should not be expected to provide information on grapheme order. As we discussed in Chapter 3, GBD and DD both seem to produce similar symptoms usually only attributable to GBD and providing suboptimal input to downstream modules may indirectly contribute to the same effect. We must therefore assume that the mechanism behind the complexity effect is located in the graphemic output buffer (GOB).

Our examination of AS's (Jonsdottir et al, 1996) raw data seemed to show a phonological effect, with AS producing many phonologically plausible errors and repair strategies akin to those suggested in Figure 6.6, for example MANY→MENY, RINSE→RINCE, and ACTUAL→ACTULE. We examined the effect of CV complexity on his error rates and from 1445 words, 105 had a simple CV structure with 50 errors (47.62%), and 1340 had a complex CV structure with 515 errors (38.43%) suggesting no significant difference between simple and complex CV error rates ($\chi^2(1) = 3.45$, $p > 0.06$). English could be the determining factor influencing this result, but there may be another. Patients LB and AS both show strong evidence for damage to the graphemic output buffer but LB is not recorded as producing phonologically plausible errors, which may in itself be very difficult in a language with an orthography as shallow as Italian. Indeed, the fact that LB showed more errors on non-words may suggest that relying on sublexical mediation was not always an option. A benefit of a phonological repair strategy is that the repair process may actually result in correct spelling; from phonological conversion of a regularly spelled word.

Since the current model does not have a phonological facility, our results can only be orthographically derived, and the clear majority of negative magnitudes in Table 6.14 indicates that complex CV words are indeed more susceptible to errors than simple-

CV words despite our corpus being English. Although both AS, and LB show clear graphemic buffer deficits, phonologically plausible errors by AS suggests possible damage in the lexical route, and a lexicality effect by LB gives rise to the possibility of damage in the sublexical route. In rationalising why orthography alone may have caused this effect in the model, we analysed the frequency of two-letter combinations in our corpus showing many more CV/VC (81%) combinations than CC/VV (21%) combinations. Tests with network A (Table 6.14), show an average magnitude difference of approximately 10 using an English corpus, and we assume that an Italian corpus would produce a more marked effect (i.e. higher magnitudes). In an Italian equivalent of Table 6.15 for example, we would expect substantially more instances of CV and VC letter combinations. We believe that this strong effect in conjunction with a lack of phonological assistance may underlie the strong orthographic effect with LB.

| Orthographic Pattern | Count | Probability |
|---|---|---|
| CC | 415 | 0.231 |
| VV | 113 | 0.063 |
| CV | 665 | 0.369 |
| VC | 607 | 0.337 |
| **Total** | 1800 | 1.000 |

**Table 6.15: Counts and probabilities for all two-letter CV patterns within our experimental corpora.**

Similar to patient AS, Kay & Hanley (1994) suggested that their English speaking patient JH was also unaffected by orthographic complexity. Italian orthography has a more regular mapping from phonology to orthography; once the regular equivalences of sound and spelling are mastered, Italian can generally be read and written correctly. Italian also has substantially more words with simple alternating CV structures than English, and geminate consonants have a highly predictable function. Kay & Hanley's definition of a simple CV word however, seems to differ from that of Caramazza & Miceli (1990) since Kay & Hanley accept any alternating CV pattern as a simple CV word (e.g. *tiger*, or colonel). Strictly speaking, only words containing an even number

of letters can be considered simple CV words. Errors for JH's simple and complex CV errors were 30/179 (16.8%), and 201/1218 (16.5%) respectively suggesting no significant difference. However, JH also produced phonologically plausible errors, in conjunction with a frequency and lexicality effect so it seems difficult to expect phonologically oriented errors to produce the same repair strategies consistent with an orthographically driven CV-complexity effect. In summary, the CV-complexity effect may be influenced by mediation through the lexical route. We now examine a method for quantifying CV complexity in words.

#### 6.4.4.1 Quantifying CV Complexity

In the previous section, we showed how orthography may affect a word's propensity for error. One possible conclusion from these results is that simple CV structures are more stable than complex CV structures. With this in mind, we now examine the behaviour of orthographic structure in the presence of damage and how this conforms to a minimum complexity principle (MCP). The MCP claims that complex CV structures become simpler as a by-product of disorder. Kay & Hanley (2004) suggested that LB's profile follows such a principle giving the examples GREMBO → GEMBO, and URLARE → ULARE. Since within-class transpositions and substitutions leave a word's CV structure unchanged, it is possible that conforming to a MCP may be a factor influencing error type, and may explain why the majority of patient errors are substitutions and transpositions. We will test whether our purely orthographic model also produces simpler CV structures in the presence of errors.

Our suggested quotient (Equation 6.10) returns a value between 0.0 and 1.0 where 0.0 suggests no CV structure whatsoever, and 1.0 suggests a complete interspersion of consonants and vowels. Simple CV words reflect an alternating CV orthography, so the quotient aims to quantify the extent of alternating CV class by subtracting the number of

letters in a word not conforming to this pattern. Therefore, CC/VV patterns reduce the quotient, and CV/VC patterns increase the quotient. Equation 6.10 counts the number of letters not comprising a CC or VV pair as a proportion of the word length where $n$ is the number of letters in the word being measured.

$$Q = \frac{n - 2(\sum CC + \sum VV)}{n} \qquad \textbf{Equation 6.10}$$

For example, 'STREETS' has the CV structure CCCVVCC. It has seven letters, and contains three CC/VV combinations (ST or TR, EE, and TS) giving a value for $Q$ of 0.143. Kay & Hanley's example errors in Table 6.16 both predict an increased quotient.

| Original Word | | Erroneous Word | |
|---|---|---|---|
| **Word** | **Q** | **Word** | **Q** |
| GREMBO | 0.333 | GEMBO | 0.600 |
| URLARE | 0.667 | ULARE | 1.000 |

**Table 6.16: Pre and post lesion quotient values for two sample words in error.**

To investigate how the quotient behaves as a result of error, we lesioned network A at various locations noting quotients for all words before and after lesioning. Table 6.17 compares how the quotients for simple and complex CV words for various word lengths either increased (/),or decreased (\) as a result of error, and in the following column, by how much (see Equation 6.11). Note that we only lesion network A here since we believe that it is the Graphemic Output Buffer (i.e. network A) which is responsible for orthographic order.

| Lesion Location | Lesion Type | Simple CV | | | | Complex CV | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 4 | % | 6 | % | 4 | % | 5 | % | 6 | % | 7 | % |
| Random | Ablate | \ | -4.0 | \ | -4.5 | / | 3.7 | / | 2.8 | / | 1.3 | / | 0.4 |
| | Noise | \ | -3.6 | \ | -4.6 | / | 2.8 | / | 2.5 | / | 0.5 | / | 0.7 |
| | Constrain | \ | -10.4 | \ | -8.9 | / | 10.4 | / | 5.9 | / | 3.3 | / | 0.1 |
| | Scale | \ | -9.1 | \ | -5.6 | / | 9.2 | / | 5.2 | / | 2.7 | \ | -0.4 |
| Input to Hidden | Ablate | \ | -5.2 | \ | -8.7 | / | 2.4 | / | 2.3 | / | 0.3 | \ | -2.1 |
| | Noise | \ | -2.5 | \ | -3.3 | / | 2.1 | / | 1.8 | / | 0.6 | \ | -0.8 |
| | Constrain | \ | -5.9 | \ | -7.5 | / | 6.0 | / | 1.8 | \ | -0.1 | \ | -1.7 |
| | Scale | \ | -7.6 | \ | -8.0 | / | 8.4 | / | 3.0 | / | 0.2 | \ | -1.5 |
| Context to Hidden | Ablate | \ | -8.6 | \ | -10.5 | / | 1.7 | / | 0.3 | \ | -1.9 | \ | -4.1 |
| | Noise | \ | -1.9 | \ | -2.5 | / | 3.0 | / | 2.0 | / | 0.7 | / | 0.1 |
| | Constrain | \ | -7.1 | \ | -7.6 | / | 3.9 | \ | -1.0 | \ | -2.5 | \ | -4.5 |
| | Scale | \ | -9.1 | \ | -9.7 | / | 5.9 | \ | -1.3 | \ | -3.1 | \ | -5.7 |
| Word identity to Hidden | Ablate | \ | -6.7 | \ | -7.6 | / | 4.5 | / | 3.9 | / | 0.5 | \ | -0.4 |
| | Noise | \ | -3.0 | \ | -4.2 | / | 2.9 | / | 2.5 | / | 0.3 | \ | -0.2 |
| | Constrain | \ | -2.7 | \ | -4.2 | / | 3.1 | / | 4.1 | / | 2.3 | / | 3.2 |
| | Scale | \ | -2.3 | \ | -3.5 | / | 2.4 | / | 3.9 | / | 2.1 | / | 3.6 |
| Hidden to Output | Ablate | \ | -3.2 | \ | -5.5 | / | 3.1 | / | 2.8 | / | 1.3 | / | 0.8 |
| | Noise | \ | -3.0 | \ | -3.9 | / | 2.3 | / | 2.2 | / | 0.3 | \ | -0.4 |
| | Constrain | \ | -8.9 | \ | -6.8 | / | 10.5 | / | 5.4 | / | 4.2 | / | 2.6 |
| | Scale | \ | -19.6 | \ | -12.5 | / | 21.2 | / | 7.9 | / | 8.0 | / | 3.0 |
| Output | Noise | \ | -4.0 | \ | -5.2 | / | 4.2 | / | 0.9 | / | 0.8 | \ | -1.2 |
| **Average** | | \ | **-6.1** | \ | **-6.4** | / | **5.4** | / | **2.8** | / | **1.0** | \ | **-0.4** |

**Table 6.17: Quotient changes for simple and complex CV words after lesioning Network A using the Moving Window context. There are no simple CV words for words with an odd number of letters. A '\' value indicates a reduction in quotient indicating more complex orthography as a result of error, and a '/' indicates an increase in quotient indicating a more simple orthography as a result of error.**

We also highlight by how much the quotient changes according to Equation 6.11.

$$\% = \frac{newQuotient - oldQuotient}{oldQuotient}$$ **Equation 6.11**

We note a consistent decrease in quotient for simple CV words. Clearly simple CV words are composed of pure CV structures and there is no way to increase the quotient. Even a single simple transposition involving a consonant and a vowel will reduce the average overall CV quotient. Complex CV words show a general quotient increase for words of four, five and six letters providing some evidence for the MCP. On longer words however, many complex errors seem to have lead to a general breakdown in CV structure thus producing lower overall quotients. Most interesting is that with very few exceptions, there is almost always a consistent quotient decrease as a function of word

length, and may indicate that an inability to retain CV structure may underlie the word length effect. Highlighted in yellow is what appears to be anomalous behaviour produced by scaling connections from hidden to output nodes, and showing much larger quotient changes than elsewhere in the table. We believe this can be explained by an extremely high number of deletes. Excessive deletes reduce the overall letter length of the word being examined, and naturally affect the quotient more than with a longer word, since the quotient is a function of the word length. For example, the error **COPE→ C** reduces the quotient from 1.0 to 0.0. Quotient decreases generally seem larger with scale and constraint techniques, and may also stem from the relatively higher number of deletes produced by these lesion types.

### 6.4.4.2 Analysis of Patient CV Complexity

Using AS's raw data, we analysed a corpus of 565 erroneous words written to dictation.

| Word Length | Increased | | | Decreased | | | Same | | | All | Simple | | Complex | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | S | C | All | S | C | All | S | C | All | | Dir | % | Dir | % |
| **4** | 0 | 18 | **18** | 2 | 7 | **9** | 2 | 8 | **10** | **37** | \ | 20.0 | / | 18.6 |
| **5** | 0 | 39 | **39** | 0 | 13 | **13** | 0 | 30 | **30** | **82** | N/A | | / | 22.6 |
| **6** | 0 | 159 | **159** | 20 | 82 | **102** | 26 | 93 | **119** | **380** | \ | 16.6 | / | 12.9 |
| **7** | 0 | 26 | **26** | 0 | 24 | **24** | 0 | 16 | **16** | **66** | N/A | | / | 4.5 |
| **Totals** | **0** | **242** | **242** | **22** | **126** | **148** | **28** | **147** | **175** | **565** | | | | |

**Table 6.18: Comparison of CV Quotient behaviour for patient AS. See text following the table for descriptions of each column heading. Values in the table refer to the number of errors.**

Table 6.18 describes the behaviour of AS's erroneous words and the effect on the quotient for each word length. Major columns labelled **Increased**, **Decreased**, and **Same** refer to the number of words with an increase, decrease or no change in quotient value respectively. The sub-columns labelled **S** and **C** refer to whether these are simple or complex CV words, and the **All** column shows the total of **S** and **C** values. The column labelled **Dir** describes the direction of the average quotient, and the **%** column specifies by how much the quotient differs from perfect spelling. These results suggest

that repair strategies of erroneous words tend to result in simpler CV structures and add evidence in favour of the Minimum Complexity Principle.

| | Complex CV | | Simple CV | |
|---|---|---|---|---|
| | **Errors** | **Within-Class** | **Errors** | **Within-Class** |
| **Substitutions** | 37.05% | 65.34% | 43.69% | 73.33% |
| **Inserts** | 19.48% | N/A | 25.24% | N/A |
| **Deletes** | 25.68% | | 23.3% | |
| **Transpositions** | 17.79% | 39.04% | 7.77% | 16.67% |

**Table 6.19: AS's error distributions grouped by simple and complex CV errors. These figures are averages of total errors across all word lengths. Within-Class errors are those where letters substitute or transpose with a letter of the same class (consonant for consonant, or vowel for vowel).**

Like the model, AS showed a greater reduction in simple CV quotients than the increase found in complex CV quotients. We consider two possible explanations for this. Firstly, the model demonstrates very high quotient decreases when applying constrain and scale lesion types, usually accompanied by considerably more deletes. Secondly, Table 6.19 details AS's very different error distributions and behaviour between simple and complex CV words. Contrary to the very high number of within-class substitutions that AS produced on words with single errors (97%), the quotient is calculated on all erroneous words, and not just a selection with single errors. Complex CV words showed many more transpositions; with many more CC/VV letter combinations, the opportunity for neighbouring transpositions is higher. For a within class transposition to occur in a simple-CV word, it must transpose with a letter at least two positions away, and long-distance transpositions are rarer than neighbouring transpositions. We believe this is further evidence to suggest that AS's *repair* strategy may have been phonologically mediated. As shown in Figure 6.6, an orthographic repair strategy might repair for example, the word THIGH as THICH substituting consonants for consonants irrespective of the resultant sound of the word. A phonological strategy however, might repair THIGH as THIOH retaining some phonological similarity with the target word.

The direction of quotient differences for AS in Table 6.18 also seems to reflect that of the model in Table 6.17. In the model, complex words almost always show a clear increase in quotient with the effect less likely in longer words, whereas AS shows a quotient increase for all letter lengths, even if only slightly in seven letter words. The model shows a progressive reduction in quotient increase as a function of word length and AS shows the same behaviour. One factor, which may have led Jonsdottir et al to consider simple CV words as susceptible to error as complex CV words, may have been their acceptance of words ending in 'Y' as having a simple CV structure. This is an arbitrary classification, and considering AS's apparent phonological errors, it seems a reasonable assumption that 'Y' is vowel-like. We therefore reanalysed AS's errors with Y defined as a vowel (Table 6.20).

| Word Length | Increased | | | Decreased | | | Same | | | All | Simple | | Complex | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | S | C | All | S | C | All | S | C | All | | Dir | % | Dir | % |
| 4 | 0 | 15 | 15 | 5 | 7 | 12 | 4 | 6 | 10 | 37 | \ | 22.2 | / | 23.4 |
| 5 | 0 | 36 | 36 | 1 | 16 | 17 | 0 | 29 | 29 | 82 | N/A | | / | 18.5 |
| 6 | 0 | 149 | 149 | 44 | 62 | 106 | 42 | 83 | 125 | 380 | \ | 21.4 | / | 15.1 |
| 7 | 0 | 25 | 25 | 0 | 25 | 25 | 0 | 16 | 16 | 66 | N/A | | / | 4.2 |
| Totals | 0 | 225 | 225 | 50 | 110 | 160 | 46 | 134 | 180 | 565 | | | | |

**Table 6.20: Comparison of CV Quotient behaviour for patient AS with 'Y' counted as a vowel. See Table 6.18 for a description of table headings.**

This assumption changes the ratio of simple CV to complex CV words from 50:515 to 97:468 almost doubling the number of simple CV words (and therefore the opportunity for simple CV errors), yet evidently making little change to the average quotient values.

We also carried out a similar analysis using data from patient BA who showed symptoms of deep dysgraphia. As BA made substantial semantic errors, and a number of non responses (i.e. no word was written) we did not include these in our analyses as they provide no value in understanding orthographic repair strategies in the presence of damage. Firstly, excluding morphological errors, semantic errors are

unlikely to have a related orthography to the word in error. Secondly a correctly spelled semantic error (e.g. YARD → LENGTH) tells us nothing about repair strategies. Thirdly, non responses have no orthographic structure.

| Word Length | Increased | | | Decreased | | | Same | | | All | Simple | | Complex | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | S | C | All | S | C | All | S | C | All | | Dir | % | Dir | % |
| 3 | 0 | 3 | 3 | 0 | 7 | 7 | 0 | 13 | 13 | 23 | N/A | | \ | 14.4 |
| 4 | 0 | 42 | 42 | 9 | 15 | 24 | 15 | 26 | 41 | 107 | \ | 20.7 | / | 39.6 |
| 5 | 0 | 81 | 81 | 0 | 45 | 45 | 0 | 32 | 32 | 158 | N/A | | / | 14.5 |
| 6 | 0 | 91 | 91 | 9 | 38 | 47 | 8 | 20 | 28 | 166 | \ | 21.2 | / | 23.1 |
| 7 | 0 | 59 | 59 | 0 | 26 | 26 | 0 | 16 | 16 | 101 | N/A | | / | 17.5 |
| 8+ | 0 | 62 | 62 | 2 | 15 | 17 | 5 | 2 | 7 | 86 | \ | 11.2 | / | 28.3 |
| Totals | 0 | 338 | 338 | 20 | 146 | 166 | 28 | 109 | 137 | 641 | | | | |

**Table 6.21: Comparison of CV Quotient behaviour for patient BA. See Table 6.18 for a description of each column.**

Our first conclusion of BA's behaviour is that the general increase in quotient value demonstrates a general tendency for orthography to simplify in the presence of damage. It is important to remember that this behaviour can only be demonstrated for complex CV words, since simple CV words will by definition have a quotient value of 1.0 and cannot simplify further. Our second conclusion is that some quotients are considerably different after damage and this may result from the high number of deletes and fragments produced by BA. In fact, where the model produces many deletes and fragments (e.g. scaling from hidden to output layers), comparable quotient changes are also produced for patient BA. Earlier in the chapter we showed that this lesion and location could produce more errors on abstract than concrete errors, as well as a frequency effect, and later in the chapter, we will show that this lesion and location also produces other symptoms shown by BA such as a serially increasing error curve. In summary, the fact that English speaking patients AS and BA show comparable behaviour to a model trained with an English corpus suggests that the model fits the behaviour of theoretically important patients.

### 6.4.4.3 Conclusions Regarding the Orthographic Effect

We have described how complex-CV words give rise to more errors than simple-CV words in the model and that a minimum complexity principle may underlie this effect. Caramazza & Miceli also describe a graphosyllabic structure grouping syllabically related graphemes in patient LB. They claim that if such a structure plays a role in processing graphemic representations, then one should expect differences in the distributions of errors for tautosyllabic and heterosyllabic clusters. Tautosyllabic is the relation between two or more letters occurring in the same syllable. In the word CONTACT for example, the first 'T' is tautosyllabic with 'A' but not with 'N'. Patient LB however made a similar percentage of errors in writing consonants in heterosyllabic (14.2%) and tautosyllabic clusters (15.2). This in itself should indicate that syllabic structure may not strongly affect erroneous letter identity. As the model has no syllabic identifier, we cannot compare heterosyllabic and tautosyllabic errors.

Patient LB was said to have made errors consistent with a graphosyllabic tier hypothesis involving adjacent letter exchanges in 13 complex words. Caramazza & Miceli for example, cite the examples SVOLTA → SVLOTA, TORNAVA → TRONAVA, which they claim demonstrate tautosyllabic transpositions. They predict therefore that the syllable *TOR* from TORNAVA is more likely to have (for example) transpositions occur within these three letters than with letters from the syllable *NA*. The transpositions for both example words are indeed tautosyllabic, each involving an adjacent consonant and vowel, and one could assume that the syllabic boundary influenced the letters involved in their transpositions. However, approaching this issue with the minimum complexity principle in mind shows that orthographic complexity may also be a factor. Using the Q value from Equation 6.10, it is apparent that for both words, the quotient associated with the erroneous output either increased, or stayed

constant: SVOLTA (Q=0.333) → SVLOTA (Q=0.677), and TORNAVA (Q=0.714) → TRONAVA (Q=0.714). So although adjacent transpositions may occur within a syllabic boundary, neighbouring transpositions may simply be more efficient than an exchange across longer serial distances.

On a further note, although our model has no graphosyllabic tier, it may be possible to provide one, such that substitutions and transpositions tend to transpose or substitute within syllables. It is beyond the scope of this thesis, but we believe that a similar mechanism to that used to identify consonant/vowel and geminate identity could provide the desired effect.

We believe that the suggested quotient provides compelling evidence in favour of a minimum complexity principle. We now investigate the behaviour of the model with respect to error distribution, serial position, and the word length effect.

### 6.4.5 Error Distribution

As described in Chapter 3, GBD patients generally make substitutions, transpositions, inserts and deletes with varying distributions. For example, in four letter words, DH (Hillis & Caramazza, 1989) makes only substitutions, whereas ML (Hillis & Caramazza 1989) and HR (Katz, 1991) make mostly deletes, and LB and JH show substitutions more than any other error type (Kay & Hanley, 1994). Deep dysgraphia patients are not known to show *typical* error distributions, although data by Katz (1991), Ward & Romani (1998), suggest a predominance of deletes, and Cipolotti et al (2004) claim that DD should lead to *type-B* GBD resulting in a majority of deletes.

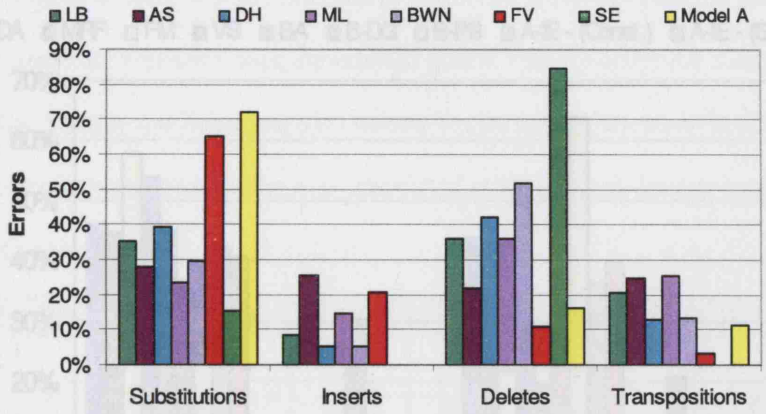



**Figure 6.7 : Error Distributions for single errors for various GOB patients and the model lesioned at the output layer using a moving window context.**

Figure 3.4 shows error distributions of various patients together with comparable performance by the model. Patient results vary, yet there is a tendency for substitutions to be most numerous followed by deletions, transpositions, and then insertions. LB for example, shows mostly **S**ubstitutions, then **D**eletions, followed by **T**ranspositions, and **I**nsertions (SDTI), whereas AS shows a pattern of SDIT, patient HE a pattern of SDTI, JH a pattern of SDIT, and the model a pattern of STDI.

The DD patients in Figure 3.5 seem to show less variance than the GBD patients in Table 6.3, and were more consistent in their error distribution, all producing at least a majority of substitutions followed by deletes. Patients DA and VS both show a pattern of SDIT, and MRF a pattern of SDTI. We also show how scaling in network A can produce behaviour similar to patient DA (Cipolotti et al, 2004). Note that constraining connections in network B with both semantic designs showed Cipolotti et al's *type-B* (patient DA) characterisation of a majority of deletes.

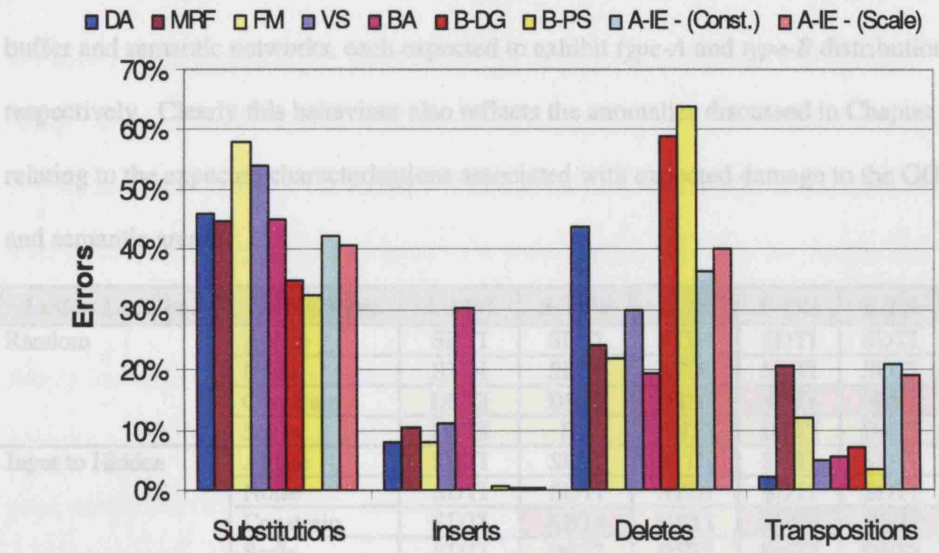



**Figure 6.8: Error distributions for single errors for many non-GOB patients and the model lesioned in network A-IE (scale and constrain word identity to hidden nodes), and network B-DG and B-PS (constraining input nodes).**

If we compare the model's behaviour with respect to lesion location, lesion type, and context type in Table 6.22, then we also note varying distributions with some lesions producing a majority of substitutions and others producing a majority of deletes. In addition to showing the overall error distributions, we colour-coded a number of cells indicating *typical type-A* and *type-B* error distributions as defined by Cipolotti et al (2004), where a majority of substitutions and deletes suggest *type-A*, and *type-B* behaviour respectively. Clear (non-coloured) cells show a distinct dominance of substitutions such as produced by GBD patients FV and JH (i.e. *type-A*). Yellow cells show a dominance of deletes giving behaviour like GBD patients ML and DH, and non-GBD patient BA (i.e. *type-B*). Orange cells indicate that the major error type is a substitution, and that the secondary error type is a delete differing by no more than 20%. This pattern reflects the behaviour of non-GBD patients such as DA VS, and MRF. As discussed in Chapter 3, *type-A* and *type-B* GBD error distributions do not fit Cipolotti et al's characterisations perfectly and it is clear from Table 6.22 that the model is able to

produce a variety of distributions after lesions have been applied to both the graphemic buffer and semantic networks, each expected to exhibit *type-A* and *type-B* distributions respectively. Clearly this behaviour also reflects the anomalies discussed in Chapter 3 relating to the expected characterisations associated with expected damage to the GOB and semantic areas.

| Lesion Location | Lesion Type | A-MW | A-POS | A-IE | B-DG | B-PS |
|---|---|---|---|---|---|---|
| Random | Ablate | SDTI | SDTI | STDI | SDTI | SDTI |
| | Noise | STDI | SDTI | STDI | SDTI | SDTI |
| | Constrain | **DSTI** | **DSTI** | **DTSI** | **SDTI** | **SDTI** |
| | Scale | **DSTI** | **DS** | **DSTI** | **DSTI** | **DSTI** |
| Input to Hidden | Ablate | SDTI | SDTI | STDI | SDTI | SDTI |
| | Noise | SDTI | SDTI | STDI | SDTI | SDTI |
| | Constrain | SDTI | **SDTI** | **DSTI** | **SDTI** | **DSTI** |
| | Scale | SDTI | **DSTI** | **DSTI** | **DSTI** | **DSTI** |
| Context to Hidden | Ablate | SDTI | SDTI | SDTI | **N/A** | |
| | Noise | SDTI | SDTI | STDI | | |
| | Constrain | SDTI | SDTI | **SDTI** | | |
| | Scale | SDTI | SDTI | **DTSI** | | |
| Word Identity to Hidden | Ablate | SDTI | SDTI | STDI | | |
| | Noise | SDTI | SDTI | STDI | | |
| | Constrain | **DSTI** | **SDTI** | **SDTI** | | |
| | Scale | **DSTI** | **DSTI** | **SDTI** | | |
| Hidden to Output | Ablate | SDTI | SDTI | STDI | SDTI | SDTI |
| | Noise | STDI | SDTI | STDI | SDTI | SDTI |
| | Constrain | **DSTI** | **DSTI** | **DSTI** | SDTI | SDTI |
| | Scale | **DS** | **DS** | **D** | SDTI | **SDTI** |
| IE | I | **N/A** | | **DTSI** | **N/A** | |
| | E | | | **DSTI** | | |
| Output | Noise | STDI | SDTI | STD | | |

**Table 6.22: Overview of Error Distribution for single errors in networks A and B.**

Glasspool et al (2006) lesioned their GSC model at network A, and network B to produce Cipolotti et al's *type-A* and *type-B* GBD behaviour respectively. Table 6.22 and Figure 3.5 show that selectively constraining and scaling weights in network A can produce both *type-A* and *type-B* error distributions. In section 6.4.2 we showed that scaling and constraining weights in network A can also produce a frequency effect similar to deep dysgraphia patients. Damaging connections from the word identity nodes may therefore have a comparable effect to lesioning network B itself.

### 6.4.6 Modification of Letter Threshold

We showed how patient error distributions can be far from consistent, and that the model also produces various error distributions depending on lesion location and type. In Chapter 5, we described the model's presentation threshold, which defined the lowest level of activation necessary for a letter to be considered a candidate for production. We now demonstrate that the model can also produce different error distributions by simply increasing or decreasing this threshold from the value originally used to train the network, and adding noise of 0.005 to the output layer of network A. The original presentation threshold of 0.80 produces the default error distribution and in Table 6.23, the threshold column lists a range of thresholds, above and below that value. The next four columns then show the resultant distribution of single errors. One might expect that an increased threshold would result in more deletes, and that a lower threshold would result in more inserts. In fact, as Table 6.23 shows, an increased threshold does result in more deletes, but a reduced threshold results in more substitutions.

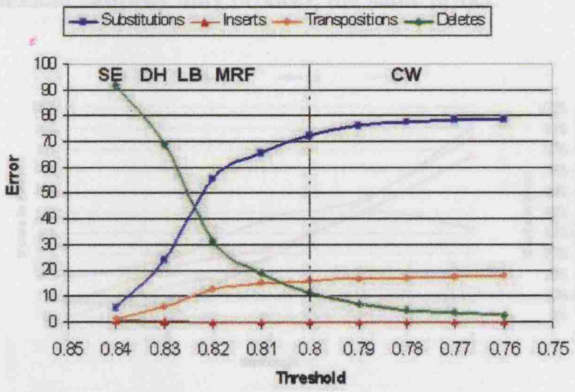
| Threshold | Substitutions | Inserts | Transpositions | Deletes |
|---|---|---|---|---|
| 0.84 | 5.714 | 0.788 | 1.480 | 92.019 |
| 0.83 | 24.202 | 0.441 | 6.258 | 69.098 |
| 0.82 | 55.745 | 0.236 | 12.625 | 31.393 |
| 0.81 | 65.734 | 0.073 | 15.119 | 19.074 |
| **0.80** | **72.302** | **0.066** | **16.327** | **11.305** |
| 0.79 | 76.108 | 0.113 | 16.531 | 7.245 |
| 0.78 | 77.688 | 0.118 | 17.300 | 4.897 |
| 0.77 | 78.353 | 0.132 | 17.733 | 3.781 |
| 0.76 | 78.706 | 0.195 | 18.130 | 2.966 |

**Table 6.23: Range of thresholds used for the presentation threshold.**

Examining the raw errors, this can be explained by being aware that substitutions increase as a direct consequence of reduced deletes. The following example should make this clear. The error LUST→[S]U[S]T→UT is produced using the standard threshold, however with a lower threshold, the same error appears as LUST→SUST. Clearly, decreasing the threshold cannot change the winning letter as it just increases the

number of (losing) letters competing. What it does do however is to allow a letter that was originally below the threshold to 'win' and therefore be presented.

A graphical interpretation of the data in Table 6.23 is shown in Figure 6.9 comparing distributions from different thresholds to those of a number of patients. Although the actual distribution percentages are unequal, they do show good qualitative matches. Clearly many patients could not be placed on this graph as the model produces very different values to those of some patients. For example, AS (SITD), and FV (SIDT) produce relatively more inserts than the model can, and patient ML (DTSI) does not match any given distribution, but qualitatively may sit somewhere on the left of the diagram. Patient SE (DS) however provided a relatively good fit producing mostly deletes and substitutions. This also applied to patients LB (DSTI) and DH (DSTI) who both produced comparable results to the model.




**Patient Distributions**

| Patient | Sub | Ins | Txp | Del |
|---|---|---|---|---|
| MRF | 44.8 | 10.3 | 20.7 | 24.1 |
| LB | 35.2 | 8.4 | 20.6 | 35.9 |
| DH | 39.5 | 5.3 | 13.2 | 42.1 |
| CW | 83.0 | 3.0 | 7.0 | 6.0 |
| ML | 23.5 | 14.8 | 25.6 | 36.2 |
| SE | 13.7 | 0.0 | 0.0 | 75.6 |

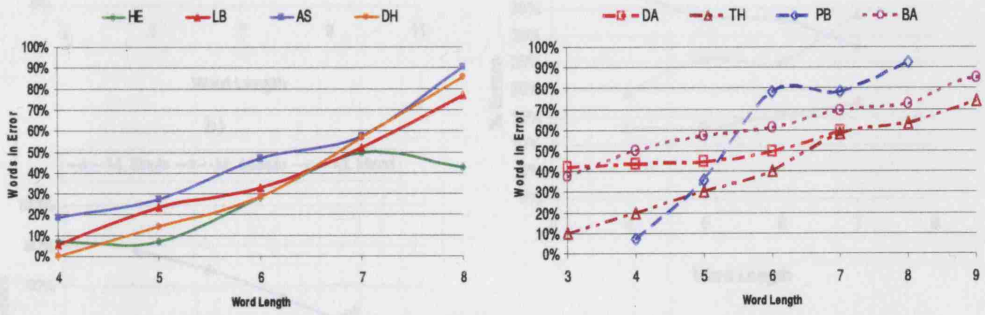
**Figure 6.9 : Resultant single error types using different recall thresholds. Patient SE, DH, LB, MRF, and CW are aligned to the threshold providing the most qualitatively similar error distribution.**

In this section, we examined how the model produces various error distributions as a result of damage. It broadly reflects the errors made by patients with an obvious deficit in producing inserts. Modifying the presentation threshold alone also produced various distributions, and there may be a case in the future to investigate whether a combination of both techniques can produce more diverse distributions in line with more patient data.

We now examine the word length effect, which is often considered a key indicator of damage to the graphemic output buffer

### 6.4.7 Word Length

It has been claimed that a word-length effect might simply exist because more letters in a word provide more opportunity for error (e.g. Posteraro et al, 1988). This may be the case, but as discussed in section 6.4.4.1, this may also be due to a general breakdown in the ability of the system to retain control of CV orthography, with shorter words providing stronger interrelationships between constituent letters. In this section, we examine the model's susceptibility to word length. The absence of a word-length effect has been held to rule out damage in the graphemic output buffer (e.g. Chialant et al., 2002, Miozzo et al., 2002) but as discussed in Chapter 3, damage situated early in the lexical pathway may produce the same effect.




(a) (b)

**Figure 6.10: Word length effect of a) four GBD patients: HE (McCloskey et al, 1994), LB (Caramazza & Miceli, 1990), AS (Jonsdottir et al, 1996) and DH (Badecker et al, 1996), and b) four non-GBD patients, DA (Cipolotti et al, 2004), TH & PB (Schiller et al, 2001) and BA (Ward & Romani, 1998).**

Figure 6.10 shows a clear word length effect for GBD patients HE, LB, AS, and DH and four patients thought to show symptoms of deep dysgraphia, DA, TH, PB, and BA. The literature normally reports the effects of word length in terms of the number of correct words decreasing as a function of word length. To remain consistent with the approach used to describe the serial position effect however, we present our results in terms of

errors. One detailed method of categorising word length errors is to group single, multiple, and mixed errors[22] (e.g. Caramazza et al, 1987). Figure 6.11 a), b), and c) show errors for patient LB, ML, and the model lesioned by adding noise to network A's output layer respectively. Qualitatively, patient behaviour tends to show a dominance of single errors in short words followed by progressively more complex errors as word length increases. As shown in Figure 6.11 c), the model provides a good qualitative match. Clearly, longer words provide a higher probability of error and with this, greater chance that these errors will be of multiple types.

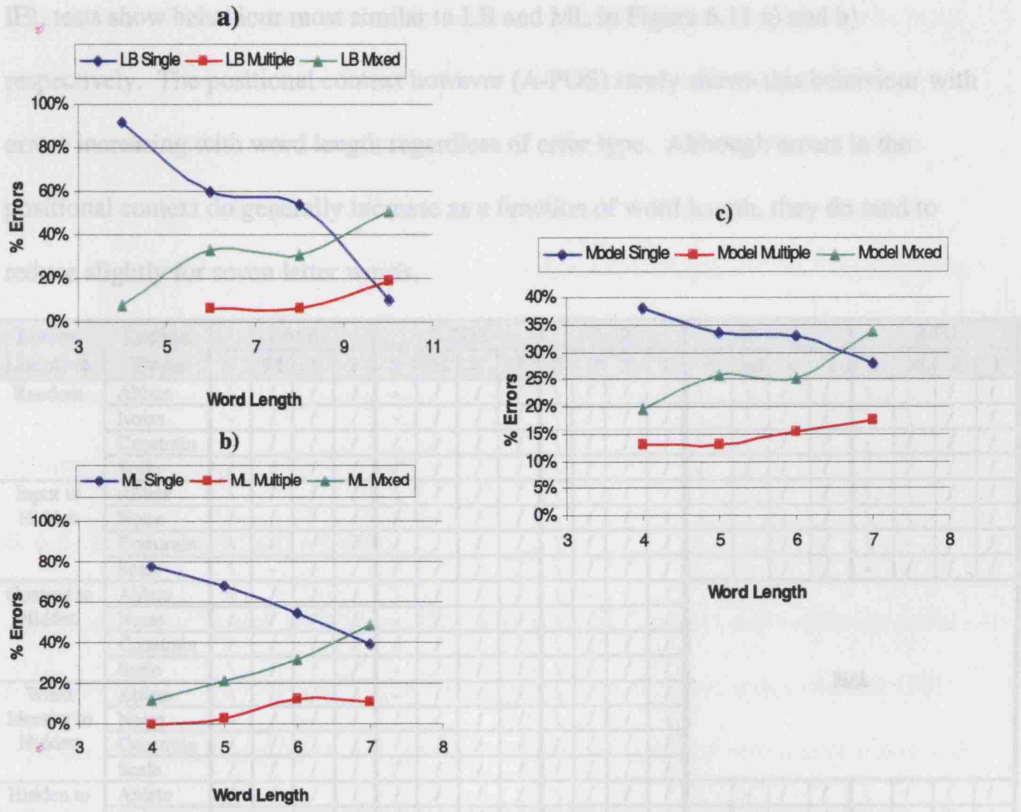



**Figure 6.11 : Normalised word length errors for single, multiple, and mixed types for a) patient LB (Caramazza et al, 1987), b) patient ML (Hillis & Caramazza, 1989), and c) lesioning network A trained with the IE context and adding noise to the output layer.**

[22] Single – word with only one error. Multiple – multiple occurrences of the same error type. Mixed – mixed occurrences of error types.

Table 6.24 shows error trends for progressively longer word lengths after lesioning the model. The sub-columns labelled **S**, **M**, **X,** and **T** represent trends for single, multiple, mixed, and total errors respectively, and the characters '/', '\', and '–' represent an increase, decrease, and no noticeable increase or decrease respectively. Our general observation is that there is an overall tendency for errors to increase as a function of word length. As with patients, the main exception to this is single errors where one might logically expect more opportunity for multiple errors as word length increases. In lesioning network A, it is clear that the moving window (A-MW) and IE contexts (A-IE), tests show behaviour most similar to LB and ML in Figure 6.11 a) and b) respectively. The positional context however (A-POS) rarely shows this behaviour with errors increasing with word length regardless of error type. Although errors in the positional context do generally increase as a function of word length, they do tend to reduce slightly for seven letter words.

| Lesion Location | Lesion Type | A-MW | | | | A-POS | | | | A-IE | | | | B-DG | | | | B-PS | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | S | M | X | T | S | M | X | T | S | M | X | T | S | M | X | T | S | M | X | T |
| Random | Ablate | \ | / | / | / | - | / | / | / | \ | / | / | / | \ | \ | / | / | \ | \ | / | / |
| | Noise | - | / | / | / | - | / | / | / | \ | / | / | / | \ | \ | / | / | \ | \ | / | / |
| | Constrain | - | / | / | / | / | / | / | / | \ | / | / | / | \ | \ | / | / | - | / | / | / |
| | Scale | / | / | / | / | / | / | / | / | \ | / | / | / | / | / | / | / | / | / | / | / |
| Input to Hidden | Ablate | \ | / | / | / | \ | / | / | / | \ | / | / | / | \ | \ | / | / | \ | \ | / | / |
| | Noise | / | / | / | / | / | / | / | / | - | / | / | / | \ | \ | / | / | \ | \ | / | / |
| | Constrain | \ | / | / | / | / | / | / | / | \ | / | / | / | \ | \ | / | / | \ | - | / | / |
| | Scale | \ | - | / | / | / | / | / | / | \ | / | / | / | \ | \ | / | / | - | / | / | / |
| Context to Hidden | Ablate | \ | / | / | / | \ | / | / | / | \ | - | / | / | N/A | | | | | | | |
| | Noise | / | / | / | / | - | / | / | / | \ | / | / | / | | | | | | | | |
| | Constrain | / | / | / | / | / | / | / | / | \ | / | / | / | | | | | | | | |
| | Scale | \ | / | / | / | - | / | / | / | \ | / | / | / | | | | | | | | |
| Word Identity to Hidden | Ablate | \ | / | / | / | - | / | / | / | \ | / | / | / | | | | | | | | |
| | Noise | - | / | / | / | / | / | / | / | \ | / | / | \ | | | | | | | | |
| | Constrain | / | / | / | / | / | / | / | / | / | / | / | / | | | | | | | | |
| | Scale | / | / | / | / | / | / | / | / | / | / | / | / | | | | | | | | |
| Hidden to Output | Ablate | / | / | / | / | / | / | / | / | \ | / | / | / | \ | - | / | / | \ | / | / | / |
| | Noise | / | / | / | / | / | / | / | / | \ | / | / | / | \ | - | / | / | \ | / | / | / |
| | Constrain | \ | - | / | / | / | / | / | / | \ | / | / | / | \ | / | / | / | / | / | / | / |
| | Scale | \ | - | - | - | / | / | - | / | / | / | / | / | / | / | / | / | / | / | / | / |
| IE | I | N/A | | | | | | | | \ | / | / | / | N/A | | | | | | | |
| | E | | | | | | | | | - | \ | \ | - | | | | | | | | |
| Output | Noise | / | / | / | / | / | / | / | / | \ | / | / | / | | | | | | | | |

**Table 6.24: Overview of the word-length effect in our model based on average errors across all lesion severities.**

This seems counterintuitive since one might expect that irrespective of serial context longer words must provide a higher probability for errors to occur. If we consider the nature of the context however, this does make sense. The A-MW, and A-IE context both introduce an element of *positional weakness* in the sense that medial positions are less stable with respect to their neighbours than at either end of the word. Glasspool & Houghton (2005) observed that the IE context is more pronounced in shorter words due to steeper gradients at either end of the word. Another view of this is that shorter words have less intra-positional ambiguity (or weakness). With the moving window (A-MW) context, adjacent letters have more overlapping context positions than letters which are positionally distant. Thus an element of ambiguity is introduced and is therefore structurally weaker as a function of word length. There is also a word length effect present when lesioning network B (columns B-DG, and B-PS). However, it is only mixed errors that for the most part show a consistent word length effect. This might be explained by the fact that with a damaged word identity being passed to the graphemic output buffer, the serial mechanism has more difficulty producing correct spelling, leading to a majority of mixed errors.

### 6.4.8 Serial position effect

As described in Chapter 3, GBD and DD patients are expected to show different serial position curves. Cipolotti et al's *type-A* and *type-B* classifications suggested that GBD patients such as LB and AS would produce a classic bow-shaped error curve where DD patients such as BA and HR would produce monotonically increasing error curves. In Chapter 5, we discussed an improved method of accounting for errors across serial position that differed from the oft-cited Wing & Baddeley (1980) approach. A full description of the method can be found in Appendix B, but in summary it provides a more balanced accountability for errors. Four letter words with Wing & Baddeley's

method for example, cannot show errors occurring in normalised position three, yet overtly biases six letter words as being more prominent in that same position three.

Figure 6.12 shows serial position curves for six patients with apparent damage to the GOB, each showing a propensity for more errors to appear in medial positions. In Appendix B we also outline a minimal descriptive notation summarising the nature of a word's serial position with curve using a four letter code. In summary, the first character describes the general shape of the curve, and the second character, the overall gradient of the curve, up, down, or none. The third character represents where the curve inflects or peaks, and the last character indicates which half of the curve contains the majority of the errors. The short codes for each patient's curve in Figure 3.6 would therefore be LB→'Λ\<<', AS→'Λ\-<', ML→'Λ\<<', DH→'Λ/>>', FM→'Λ\->', and BH→'Λ\>>'. The major difference between the first and last three patients generally relates to which half of the error-curve most errors appear in.

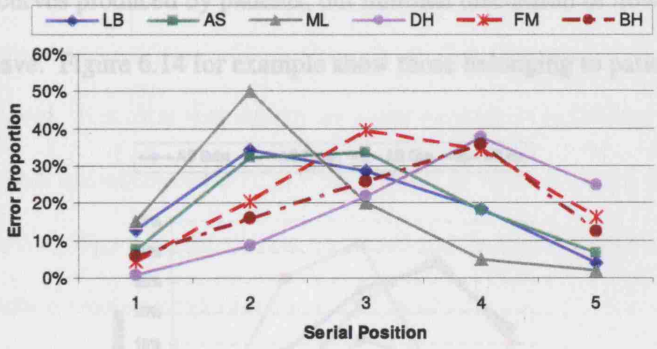



**Figure 6.12 : Normalised single error positions for six patients exhibiting damage to the GOB – LB (Caramazza & Miceli, 1990), AS (Jonsdottir et al, 1996), ML and DH, (Hillis & Caramazza, 1989), FM (Tanturier & Rapp, 2004), and BH (Sage & Ellis, 2004).**

Short codes for each *type-B* patient curve in Figure 6.13 are, HR→'Λ/>>', BA→'//--', DA→'//--', PB→'//--' and TH→'//--'. Patients BA, DA, PB, and TH show the

expected serially increasing curves, whereas HR strictly shows a bow shaped curve that is qualitatively serially increasing like the other *type-B* patients.

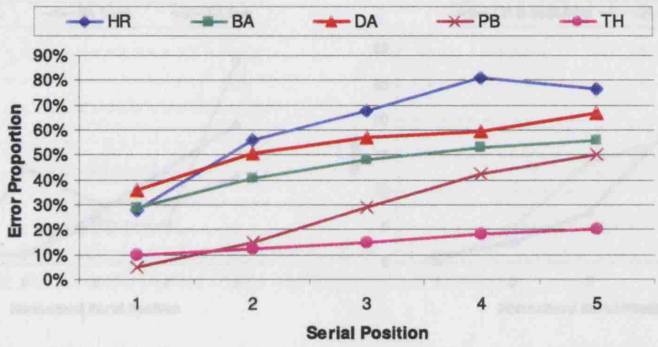



**Figure 6.13: Normalised serial position errors for five patients with *type-B* serial curves HR (Katz, 1991), BA (Ward & Romani, 1996), DA (Cipolotti et al, 2004), PB & TH (Schiller et al, 2001).**

We now investigate how constituent errors behave with respect to serial position.

### 6.4.8.1 Error Type behaviour across Serial Position

As discussed in Chapter 3, there are many references in the literature to the overall serial position curves produced by patients, but minimal discussion of how constituent error types behave. Figure 6.14 for example show those belonging to patient AS.

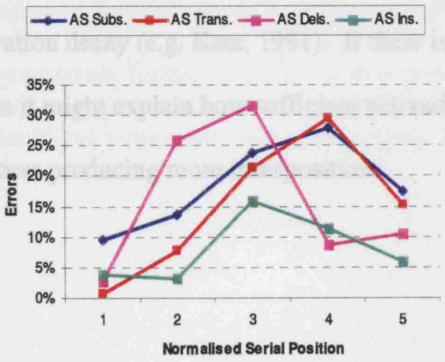
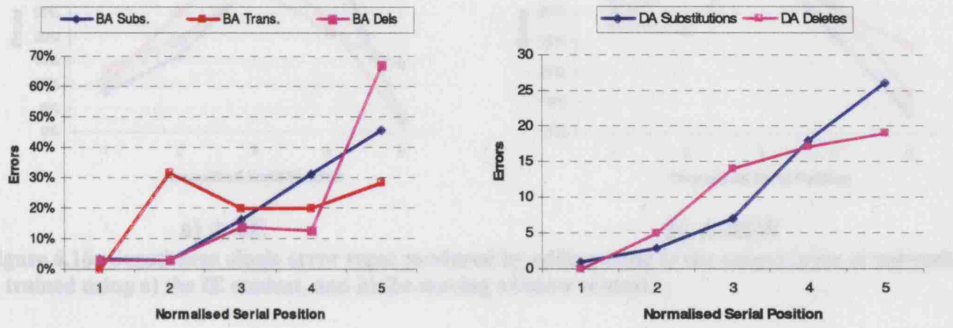



**Figure 6.14: Constituent error types for *type-A* GBD patient AS.**

Transpositions and substitutions seem to show very similar curves. Section 6.4.3.3 discussed the effect of CV status on both of these types so it should not be surprising for

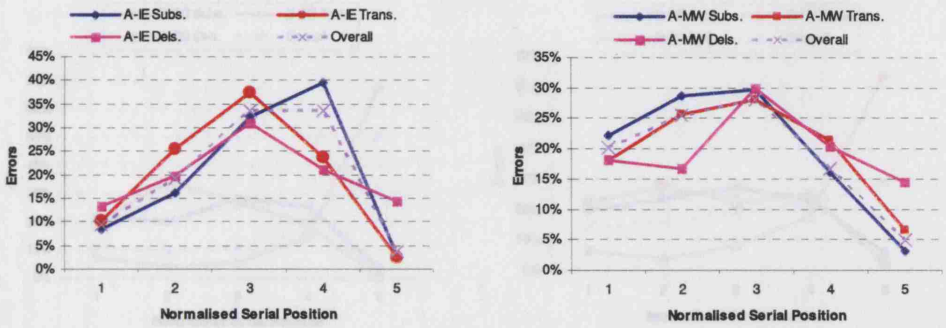
these same error types to show similar serial position curves. Deletes and inserts are however, very different showing a dominance of errors in different serial positions.




**a) Patient BA** **b) patient DA**

**Figure 6.15: Constituent single error types for two *type-B* GBD patients, BA, and DA.**

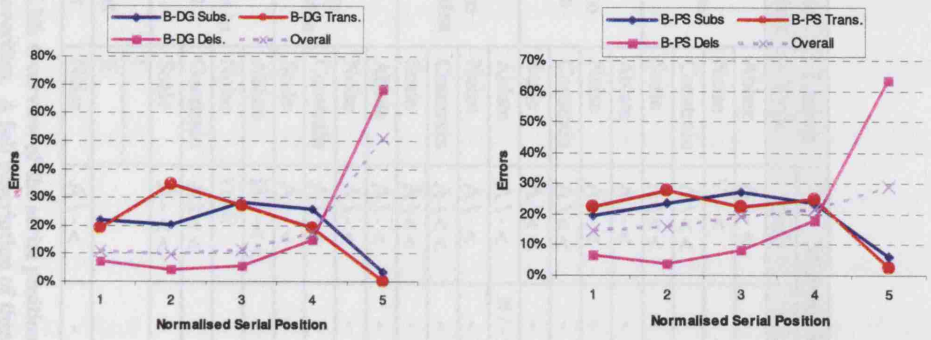
Figure 6.15 shows the individual error types contributing to the overall serial positions for patients BA and DA. Both patients show relatively consistent increases in deletes and substitutions across serial position, yet transpositions do not progressively increase for BA. Later in the chapter we examine a new lesion technique producing *type-B* serial behaviour where transpositions behave similarly. Deletes result from an overall lack of activation whereas transpositions indicate that activation is present, but not sufficiently for the correct letter. It is clear that deletes are more prominent in trailing positions suggesting possible activation decay (e.g. Katz, 1991). If there is indeed a decay process taking place, then it might explain how sufficient activation may exist early in the letter production process producing more transpositions.




**a) A-IE** **b) A-MW**

**Figure 6.16: Constituent single error types produced by adding noise to the output layer of network A trained using a) the IE context, and b) the moving window context.**

As with previous tests using the model, we assume that the lesions to network A and network B should produce *type-A* and *type-B* GBD symptoms respectively. Figure 6.16 shows constituent serial position curves resulting from noise applied to the output layer of network A trained with the a) IE and b) moving window (A-MW) contexts, each producing a clear bow-shaped effect. The moving window (A-MW) context seems to produce errors earlier than the IE context and this may be explained by the fact that the IE context produces very high gradients at the ends of words resulting in a high level of accuracy at these points. This does not seem to be as pronounced with the moving window context. Both examples however show a considerable decrease in errors toward the end of the word. As we explain further, this may be due to the inhibition mechanism reducing the number of candidate letters available to participate in erroneous activity.




**a) B-DG** **b) B-PS**

**Figure 6.17: Constituent single error types produced by scaling weights from the hidden to output layer of network using a) Glasspool semantic design, and b) Plaut & Shallice vector design.**

Comparing the results in Figure 6.17 to those of BA and DA, the model, like the patients clearly shows progressively more deletes as a function of serial position. As summarised in Chapter 3, patients DA, BA, HR, TH, and PB all make progressively more errors as a function of serial position, and the overall error rate of the model seems to reflect these patients well, although substitutions do not consistently seem to increase as a function of serial position.

Figure 6.16 and Figure 6.17 *graphically* demonstrated the model's performance. We now show the serial position effect for all three contexts on network A (Table 6.25) and both semantic designs on network B (Table 6.26) using our short form descriptive notations (see Appendix B).

| Lesion Location | Lesion Type | Moving Window Context (A-MW) | | | | | IE Context (A-IE) | | | | | Positional Context (A-POS) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Sub | Ins | Txp | Del | Tot | Sub | Ins | Txp | Del | Tot | Sub | Ins | Txp | Del | Tot |
| Random | Ablate | Λ\-< | - | Λ\-< | И/-> | Λ\-< | И\-< | U\>> | Λ\-< | W/-> | И\>> | \\-< | N/-> | Λ\-< | //-> | И\<< |
| | Noise | \\-< | - | Λ\-< | //-> | И\-< | Λ\>> | \\<< | Λ\-< | N/>> | Λ\>> | \\-< | - | Λ\-< | //<> | \\-< |
| | Constrain | Λ\<< | - | Λ\-< | //-> | //-> | И\>> | - | Λ\-< | //-> | //-> | \\-< | - | Λ\-< | U/<> | U/-> |
| | Scale | Λ\<< | - | И\-< | //-> | //-> | Λ\>> | - | Λ\<< | //-> | //-> | \\-< | - | - | U/-> | U/-> |
| Input to Hidden | Ablate | Λ\-< | - | Λ\-< | //-> | Λ\-< | Λ\>< | \\<< | Λ\-< | N/>> | Λ\>> | \\-< | - | Λ\-< | //-> | \\-< |
| | Noise | Λ\-< | - | Λ\-< | //-> | Λ\-< | Λ\>> | - | Λ\-> | N/>> | Λ\>> | \\-< | - | Λ\-< | //-> | \\-< |
| | Constrain | Λ\<< | - | Λ\-< | //-> | N/>> | И\>> | - | Λ\-< | //-> | //-> | \\-< | - | Λ/-> | U/-> | U/-> |
| | Scale | Λ\<< | - | Λ\-> | //-> | N/>> | И\>> | - | Λ\<< | //-> | //-> | \\-< | - | //-> | U/-> | U/-> |
| Context to Hidden | Ablate | Λ\-< | N/-> | Λ\-< | //-> | Λ\-< | Λ\>> | N/>> | Λ\-< | //-> | Λ/>> | \\-< | N/-> | Λ--< | //-> | Λ\-< |
| | Noise | Λ\-< | - | Λ\-< | //-> | Λ\-< | Λ\>> | \\<< | Λ\-< | N/>> | Λ\>> | \\-< | N--> | Λ\-< | //-> | \\-< |
| | Constrain | Λ\<< | - | Λ\-< | N/>> | Λ\-< | Λ\>> | - | Λ\-< | //-> | Λ/>> | Λ\<< | - | Λ/-> | //-> | Λ\<< |
| | Scale | Λ\<< | - | Λ\-< | N/>> | Λ\-< | Λ\>> | - | Λ\<< | //-> | Λ/>> | \\-< | - | Λ/>> | N/-> | \\-< |
| Word Identity to Hidden | Ablate | Λ\-< | - | Λ\-< | //-> | Λ\-< | Λ\>> | Λ\<< | Λ\<< | N/>> | Λ->> | \\-< | //>> | Λ--< | N/<> | \\-< |
| | Noise | Λ\-< | - | Λ\-< | //-> | Λ\-< | Λ\>> | Λ\<< | Λ\-< | N/>> | Λ->> | \\-< | - | Λ\-< | //-> | \\-< |
| | Constrain | Λ\-< | - | Λ\<< | //-> | //-> | Λ->> | - | Λ\-> | //-> | //-> | Λ\<< | - | Λ/-> | //-> | N/<> |
| | Scale | Λ\-< | - | Λ\<< | //-> | //-> | И->> | - | Λ\-< | //-> | //-> | Λ\<< | - | Λ/-> | //-> | N/<> |
| Hidden to Output | Ablate | И\-<< | - | Λ\-< | U/<> | И\<< | И\>> | W/-> | Λ\-< | N/>> | Λ\>> | \\-< | N/-> | Λ--< | //-> | И\<< |
| | Noise | \\-< | - | Λ\-< | U/<> | \\-< | Λ\>< | \\<< | Λ\-< | N/>> | Λ\-< | \\-< | - | Λ\-< | //-> | \\-< |
| | Constrain | Λ\<< | - | Λ\<< | U/<> | U/-> | Λ\>< | - | Λ\-< | U/-> | //-> | Λ\<< | - | Λ-<< | //-> | U/-> |
| | Scale | Λ-<< | - | - | U/<> | U/<> | - | - | - | W/-> | W/-> | Λ-<< | - | - | U/-> | U/-> |
| I/E Context | I | N/A | | | | | \\-< | U/>> | \\-< | W\-< | \\-< | N/A | | | | |
| | E | | | | | | Λ/>> | //-> | //-> | //>> | //-> | | | | | |
| Output | Noise | Λ\-< | - | Λ\-< | И-->> | Λ\-< | Λ\>> | - | Λ\-< | Λ--> | Λ\>> | Λ\<< | - | Λ\-< | И-<< | \\-< |

**Table 6.25: Overview of the serial position effect in network A with the moving window, Initiator/End, and Positional contexts. Results are averages of single errors across all lesion severities. A full description of these descriptive codes can be found in Appendix B.**

The results in Table 6.25 suggest that context does affect serial behaviour for network A. Firstly, the typical total error curves produced by both moving window and IE contexts are bow shaped. In instances where this is not the case, they usually increase serially and are due to constrain and scale lesion types. As discussed earlier, increasing serial position curves may be caused by activation decay, and both constrain and lesion types have an effect of reducing the overall contribution to nodes with a consequent reduction in activation value. The positional context does not provide an overall bow-shaped curve, which is expected since every serial position is considered to be equally prone to error. Rather than a flat serial position curve however, the most common curve was serially decreasing similar to right-side neglect. As we now explain, we believe this can be accounted for by the inhibitory effect. Table 6.22 showed that the majority of errors were substitutions, and as serial position increases, there is a concomitant reduction in candidate letters available for substitution. In order to rationalise this behaviour, it is important to review the inhibition algorithm first described in chapter 5. During recall, the letter produced is inhibited to prevent its immediate reoccurrence where inhibition is absolute in the position following production, and progressively less inhibitory as the distance from production increases. During training however, the letter that *should* have been produced is inhibited in the same fashion. This *self-editing* approach (Glasspool, 1998) ensures that erroneous letters produced early in the word are not excluded from being produced correctly later due to erroneous inhibition. Thus at the beginning of the word every letter is a candidate for erroneous substitution whereas fewer letters qualify, as serial position increases. Letters already produced will still be slightly inhibited, and letters erroneously produced during the training process are also likely to have relatively lower activations than letters not participating in the training process. If we ignore the

inhibitory effect, we could consider every letter as a likely candidate for error and that the serial position curve would be qualitatively flat.

With the moving window and IE contexts, there is also a difference in where errors tend to be produced. The IE context tends to produce more errors towards the end of the word, whereas the moving window context tends to produce them earlier. Where they are consistent is once again for constrain and scale lesion types. As mentioned earlier, the effect of reduced activation may produce a decaying effect. This is supported by Table 6.22 showing that these lesion types produce a majority of deletes. The model does tend to produce qualitatively similar errors to those made by patients. Patient AS's substitutions were (Λ/>>). With slightly more errors in final positions, the IE context would provide a very good match to these figures for most lesion locations. Similarly, AS's transpositions (Λ/>>) would also provide a good match were there more trailing errors. A revised inhibition algorithm may produce results more in line with patient errors. Deletes for AS (N/><) are similar to a number of scale and constrain lesion locations in the IE context but for the fact that most errors for AS occur early in the word and not later. The model unfortunately does not produce sufficient inserts to make a valid comparison.

To examine the effect of semantic design on serial position, we used the output of a lesioned network B as input to an intact network A trained with the moving window context. Table 6.26 shows results for both semantic vector designs suggesting that despite results between semantic features design showing slight differences, results are in the main quite similar. Both designs show regular increases in deletes shown by patients BA and DA. Both designs show bow shaped curves for transpositions and though this does not perfectly reflect behaviour for BA, a slight increase in trailing errors would (as with lesions to network A), provide a better fit. Both BA and DA also show a serial

increase in substitutions. It is clear from Table 6.22 that the lesion strategies applied to network B did not produce a majority of deletes in sufficient instances to affect the level of activation passed to network A. In any case, we believe that the inhibition mechanism used in network A makes progressively increasing substitutions highly unlikely without resorting to another approach. As with network A the quantity of inserts produced by the model is almost non-existent, but rather than lose information we have provided statistics based on what was produced. Qualitatively, they are as effectively zero.

| Lesion Location | Lesion Type | B-DG | | | | | B-PS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Sub | Ins | Txp | Del | Tot | Sub | Ins | Txp | Del | Tot |
| Random | Ablate | Λ\-< | - | Λ\-> | //-> | N/>> | Λ\-< | - | Λ\-< | //-> | N/>> |
| | Noise | Λ\-< | - | Λ--< | //-> | N/>> | Λ\-< | //>> | Λ\-< | //-> | Λ/-> |
| | Constrain | Λ\-< | - | Λ\-< | //-> | N/>> | Λ\-< | - | Λ--< | //-> | //-> |
| | Scale | И\-< | - | Λ\<< | //-> | //-> | Λ\-< | N->> | Λ\<< | //-> | //-> |
| Input to Hidden | Ablate | Λ\-< | - | Λ\<< | //-> | N/>> | Λ\-< | //>> | Λ\-> | //-> | //-> |
| | Noise | Λ\-< | - | Λ--< | //-> | N/>> | Λ\-< | И\-< | Λ\-< | //-> | N/-> |
| | Constrain | Λ\-< | - | Λ\<< | //-> | N/>> | Λ\-< | И--> | Λ\-< | //-> | //-> |
| | Scale | Λ\-< | - | Λ\-< | //-> | //-> | Λ\<< | - | Λ-<< | //-> | //-> |
| Hidden to Output | Ablate | Λ\-< | \\<< | Λ--< | //-> | Λ/-< | Λ\-< | - | Λ\-< | //-> | Λ/-> |
| | Noise | Λ\-< | N/-> | Λ\-< | //-> | Λ\-< | Λ\-< | - | Λ\-< | //-> | Λ/-> |
| | Constrain | Λ\-< | - | Λ\-< | //-> | N/>> | Λ\-< | N/>> | Λ\<< | //-> | N/>> |
| | Scale | Λ\-< | - | Λ\<< | //-> | N/>> | Λ\-< | - | M\-< | //-> | //-> |

**Table 6.26: Overview of serial position effect in network B for Glasspool vectors (B-DG), and Plaut & Shallice vectors (B-PS). Results are averages of single errors across all lesion severities. A full description of these descriptive codes can be found in Appendix B.**

At a general level, we examined how lesioning network A and network B produce what amount to Cipolotti et al's (2004) *type-A*, and *type-B*, serial position curves. Although either network can under the right circumstances produce both bow-shaped, and serially increasing serial position curves, we believe that network A is *predisposed* to producing bow-shaped serial position curves (i.e. *type-A*), and network B to producing increasing serial position curves (*type-B*). In other words, despite our tests being more rigorous than those used by Cipolotti et al (2004) our results still reflect their observations that locus of impairment can affect the type of serial position curve produced by a patient, at least insofar as the model of the GOB and semantic systems are concerned. Our first

conclusion therefore is that the model appears to do what is expected. Our second conclusion is that it does not do so in every detail. As discussed, substitutions produced by lesioning network B do not generally produce *type-B* serial position curves.

An objective of a serial position context in network A is to introduce a level of instability in medial serial positions. The fact that the positional context does not produce this and the moving window and IE contexts do, verifies that such mechanisms are necessary to fulfil appropriate serial position curves. It also shows that these mechanisms behave in line with patient errors across a wide range of locations and severities. We also noted serial position effects similar to those found in right-side neglect, but as discussed, we believe this was an artefact of the inhibitory mechanism. We now investigate a specific approach to introducing both left and right oriented serial position errors approximating aspects of left and right-side neglect.

### 6.4.9 A context specific lesion approach

In the previous section we examined the effect of errors on serial positions, where *type-A* behaviour corresponded to the classic bow-shaped curve, and *type-B* behaviour produced a steady increase in errors as a function of serial position. Errors dominant on a single side are also one symptom of unilateral neglect, and although neglect as a disorder manifests a much broader range of symptoms, it conveniently produces a property, which the GSC model has so far not been shown to produce; namely left-side neglect. Despite being able to simulate right side neglect producing *type-B* symptoms, Ward (1997) found left neglect more difficult. He simulated symptoms of right side neglect by omitting the 'E' component of the IE context producing noticeable errors after the first two to three letters, but removing the I component did not produce a complementary right-side effect due to an overall lack of activation at the letter layer. We tried a similar approach to Ward by adding noise and scaling IE values. However, we were less

successful than Ward. This may have been due to the fact that the neglect-like symptoms were not consistent over lesion severities

In order to provide a lesion method that consistently addresses both left and right neglect, we will assume that one should be able to lesion either the I or E component in isolation producing left and right neglect errors respectively. To achieve this, we have modified the standard IE equations (Equation 6.12 and Equation 6.13) first described in Chapter 5 to include a localised noise element.

$$I = F^{p} \qquad \textbf{Equation 6.12}$$

$$E = F^{L-p} \qquad \textbf{Equation 6.13}$$

Compared to the standard IE equations above, Equation 5.1 and Equation 6.15 include a component, which is more effective earlier in the process for the I curve, and more effective later in the process for the E curve. Lesioning the I curve has most effect at the beginning of a word, and lesioning the E curve, has most effect at the end of a word.

$$I' = F^{p+\Delta^{p}} \qquad \textbf{Equation 6.14}$$

$$E' = F^{L-p+\Delta^{(L-p)}} \qquad \textbf{Equation 6.15}$$

Where $\Delta$ represents the lesion severity, and thus its tendency to diverge from the true I or E function. Figure 6.18 a) and b) below show the original I and E curves and three additional I' and E' curves with progressively increasing values of $\Delta$ (0.50, 0.75, 1.00).

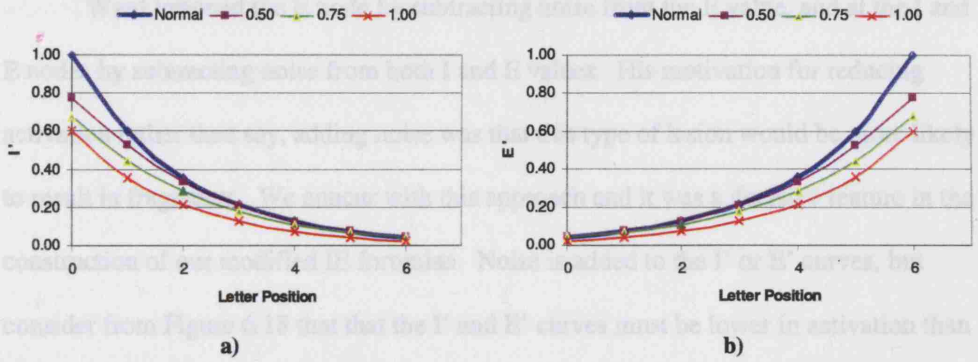



**Figure 6.18: Range of I' and E' lesions compared to the original IE curves. Curve difference is due to various lesion values of $\Delta$**

With larger values of $\Delta$, the difference between the original and modified curve is more pronounced across the entire word length. With smaller values of $\Delta$, the difference between the original and modified curve tends to show only at the beginning or end of the I and E curves respectively. This approach thus fulfils our objective of ensuring that lesioning is both predictable and that it only affects the region of the word intended. A further objective of our lesion approach is that it suits the testing of quasi-subjects, with random seeds providing different results for each subject. We expect varying errors in the same way that patients have been known to provide different responses to the same stimulus. Patient JH (Kay & Hanley, 1994) for example writes the correct form of the word *offerings* elsewhere in his text before he misspells it as *offerrerings*. McCloskey et al (1994), also note this occurring with patient HE, where of 174 incorrectly spelled words, 97% of these were also spelled correctly on at least one occasion, and 54% were spelled correctly at least twice and incorrectly at least twice. We therefore introduce a further modification to the lesion type by adding noise to our modified IE curves. This allows subtly different results and is arguably more neurologically plausible. Figure 6.19 shows an I' curve with the shaded area in yellow indicating possible erroneous values resulting from noise. The corresponding E' curve would be its mirror image.

Ward lesioned the E node by subtracting noise from the E value, and at the I and E nodes by subtracting noise from both I and E values. His motivation for reducing activation rather than say, adding noise was that this type of lesion would be more likely to result in fragments. We concur with this approach and it was a decisive feature in the construction of our modified IE formulae. Noise is added to the I' or E' curves, but consider from Figure 6.18 that that the I' and E' curves must be lower in activation than the original I and E curves. To ensure that adding noise does not exceed the original I or E values, it is therefore critical that range of noise lies between $\pm(I - I')$ or $\pm(E - E')$ for each position, or a reduction in activation cannot be guaranteed.

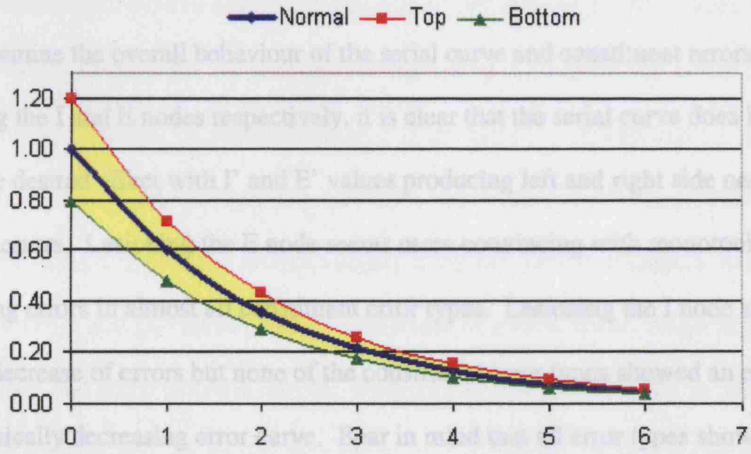



**Figure 6.19: An I' curve showing the range of potential values after applying up to 20% of noise.**

Ward (1997) claimed that by modifying sequencing nodes, it would not be possible to provide out-of-word intrusions such as *pencil* → *pancil*. Rather only perseverations would be possible (e.g. *pencil* → *pepncil*) or anticipations (e.g. pencil → *peincil*). The rationale behind this claim seems to be based on the fact that sequencing nodes only activate letter identities comprising the word, with other letters being less active. This is clearly not the case with our model as the lazy update algorithm allows a non participatory (out-of-word) letter's activation to retain any value as long as it does not

exceed the presentation threshold. In the presence of lesions, it is highly likely that letters near the presentation threshold are produced resulting in many out of word substitutions and inserts. Out of word letters are also common with patients. In our analysis of patient AS, his substitution errors included out of word letters 51% of the time, and inserts 51% of the time. Patient BA showed an even more pronounced out-of-word effect with substitutions (72%), and inserts (82%).

| Error Type | Lesion Type | Initiator/End Context | | | | |
|---|---|---|---|---|---|---|
| | | Substitutes | Inserts | Deletes | Transpositions | Total |
| Total Errors | I' | Λ\<< | N//< | U\<< | Λ\<< | \\-< |
| | E' | Λ/>> | //>> | //>> | Λ/>> | //-> |
| Single Errors | I' | \\<< | (negligible) | V\>< | \\<< | \\<< |
| | E' | Λ/>> | N/>> | //>> | //-> | //-> |

**Table 6.27: Overview of serial effect in network A after lesioning I and E nodes**

If we examine the overall behaviour of the serial curve and constituent errors after lesioning the I and E nodes respectively, it is clear that the serial curve does indeed show the desired effect with I' and E' values producing left and right side neglect spelling errors. Lesioning the E node seems more convincing with monotonically increasing errors in almost all constituent error types. Lesioning the I node shows an overall decrease of errors but none of the constituent error types showed an expected monotonically decreasing error curve. Bear in mind that all error types showed more errors early in the word than later in the word, and we would therefore argue that the lesion type works as expected. We argue that although the inhibitory mechanism may be responsible for some left-side neglect symptoms, this lesion approach is designed to produce neglect-like symptoms, such that they cannot be considered a simple artefact of the model's natural function.

## 6.5 Discussion

The main focus of our investigations in this chapter has been to validate the performance of our extended model against models discussed in Glasspool (1998), Houghton et al (1994), Glasspool & Houghton (2005), Glasspool et al (2006), and expand on the their analyses now possible with access to more computing power than was available at the time for many of these publications. One major difference in our approach from the GSC model is our use of multiple lesion severities, quasi-subjects, multiple corpora, and various semantic vector designs. We believe these techniques expose the model to more rigorous testing. The fact that a more extensive battery of tests produces qualitatively comparable results shows that the model is generally robust in the presence of damage. We also believe that using the augmented model has allowed us to examine a number of questions related to the nature of patient results, namely the influence of orthographic complexity on errors and the minimum complexity principle.

We first compared the model's ability to reflect patient behaviour with respect to aspects of GBD and DD manifested by symptoms of concreteness, frequency, the word-length effect and serial position curves. Despite some initial difficulty in establishing a metric for significance, we arrived at a measure of difference, which we believe is appropriate for our comparisons insofar as it quantifies how significant and substantial the differences are. Our initial results were similar to those of the GSC model, with lesions applied to network A or B producing *type-A* and *type-B* behaviour respectively.

We then investigated the dynamics of consonants and vowels in the presence of lesioning. Adding CV nodes clearly caused a significant change in error behaviour with consonants and vowels tending to transpose and substitute within class more than without CV nodes. Similar behaviour is well documented in Glasspool et al (2006) as well as in other work by Glasspool & Houghton (2005). An interesting contribution

however relates to CV complexity in defining a quotient for CV complexity, making it possible to analyse orthographic behaviour in the presence of damage. There were two motivations behind the use of the quotient. The first was to substantiate whether error behaviour was measurably affected by a minimum complexity principle. The evidence suggests that this is indeed the case both in the model and in patient data, and we believe that this may be a factor in why GBD patients produce a majority of substitutions and transpositions as these will have minimal affect on CV complexity. The second motivation was to substantiate whether CV structure is a factor in determining a word's propensity for error. Caramazza & Miceli (1990) claim that complex CV words are more prone to error, whereas Jonsdottir et al (1996) claim that the effect is strongly influenced by language, with Italian more likely to produce the effect. The English patient AS did not seem to be as susceptible to CV complexity, but we believe that this can be explained by a strong phonological effect on AS's repair strategies. Our model trained on an English corpus with no phonological mechanism was able to mirror the qualitative nature of Italian patient LB's results.

We then examined whether the model could produce varying types of error distributions as a consequence of damage. For the most part, the model produced results broadly reflecting those of a number of patients. Modifying the letter threshold also had an effect on error distributions. Although a broad range of distributions was produced through standard lesioning, it was clear that error distributions of at least five patients could be explained to some extent by modifying the letter threshold alone.

In examining the serial effect the choice of serial context mechanism also seemed to make a difference. The moving window and IE contexts should by design produce bow-shaped error curves whereas the positional context is not expected to. This was indeed the case but an unexpected effect of decreasing errors across all serial positions

was noted. We believe that the inhibitory mechanism causes a subtle reduction in errors as serial position increases and future work may highlight whether this is indeed the source of the effect. We then investigated a specific lesion technique, which can produce the effects of serially increasing and decreasing errors as a function of serial position.

### 6.5.1 Faults with the model

The model appears to produce many aspects of Cipolotti et al's *type-A* and *type-B* GBD properties, yet there seem to be some issues that must be addressed for the model to be more representative of patient behaviour. Firstly, the model does not accurately cater for the broader range of error distributions present in patient data. We showed that by altering the presentation threshold, it is possible to provide various combinations of substitutions, deletes, and transpositions. There is still a clear deficiency in producing erroneous inserts, and even modifying the model's threshold did not help. Glasspool & Houghton (2005) describe a technique for producing more inserts that relies on modifying the competitive filter. Normally, the most active letter exceeding the presentation threshold is output, and where no letter exceeds that threshold, nothing is output resulting in an omission. They argue that when two letters are both very highly active, and have sufficiently close activations to both be considered *winners* that they could both be presented in the same time slice. Their modification seemed to be partially successful with inserts far more likely for shorter words. At some future date, it may be worth investigating how a similar approach can be used with our augmented model.

As discussed earlier, the design of the semantic feature vectors in network B is aimed at differentiating words on the basis of concreteness. More concrete words are therefore given correspondingly more attributes making them more robust in the presence of damage. Network B seems to play the role of both orthographic lexicon and

semantic system and a future incarnation of the model may consider separating these. One might argue that in a three-layer model, the input layer (semantics) converted to the output layer (lexical-identity) suffice as a simple model for both. However, we believe that this would be difficult to defend as the scope of each module expands to cater for more complex behaviour. For more plausible semantic errors for example, one might consider that damage to the semantic system should not just result in a concreteness effect, but also produce semantically related words in the presence of damage. Hinton & Shallice (1991) for example used the concept of basins of attraction within a semantic space where active attributes actually described the nature of the word (e.g. *has-legs*, *found-on-farms*, *does-fly*), and conceptually similar words would have similar features (e.g. *chicken*, and *duck*). In principle, we believe that with such a design, the GSC model should be able to produce semantic errors. A simple test using Plaut & Shallice's (1993) corpus of forty words and their semantic design (see Appendix F) produced at least one semantic error (LOAN → PLA[N] → PLA). The attributes for LOAN and PLAN are strongly correlated ($\rho = 0.467$, N=98, $p < 0.01$) suggesting that the spelling error has a semantic link. Similar semantic/orthographic errors were shown by FM (Tainturier & Caramazza, 1996), TH (Schiller et al, 2001), and BA (Ward & Romani, 1998). In FM's case, the response ELEPHANT → ROTIGGE was rationalised by the authors as a two step process: ELEPHANT → GIRAFFE → ROTIGGE with the semantic error followed by consistent within-class substitutions. TH made a similar error (ARTICHOKE → CAULFLOWER), as did BA (PUDDING → DESSET, SPAIN → MEX, CABINET → CUPBROAD). In our example, the semantic error was followed by a single delete. In extending the model at a future date, modifying the semantic design to incorporate corpus characteristics may provide a broader range of patient related symptoms.

One possible critique of the model rests with the connectionist design of network B. We have already stated that Glasspool placed no theoretical weight on the network itself as it was considered a proxy for providing input to network A that had a predictable behaviour with respect to concreteness and frequency. We believe that further investigation may be necessary to produce a more robust semantic system if further research involving the relationship between the graphemic buffer and the lexical-semantic disorders is planned, and we provide a possible design in Chapter 8.

In analysing serial position errors, the moving window and IE contexts produced clear bow-shaped error curves similar to those of GBD patients, and it was predicted that the positional context would produce very different behaviour. In fact contrary to a consistent error rate across serial position, a steadily decreasing error was produced consistent with aspects of left-side neglect. We concluded that the positional context exposed an underlying weakness in the model. The inhibitory mechanism used to suppress double letters had an indirect effect of suppressing potential candidates for substitutions (Table 6.25). With the moving window (A-MW), and IE contexts, the effect was obfuscated by the bow-shaped curve but on closer examination may be evident in the moving window context with more errors typically located towards the beginning of words. Clearly, this is an hypothesis and further investigation would be necessary to substantiate that an alternative inhibition mechanism, would not produce the same effect. Indeed, allowing more candidate letters to participate in the competitive process for successive letter positions, and at higher activations may even produce more erroneous inserts.

### 6.5.2 Comparison with GSC model

Glasspool et al (2006) have described limitations in the GSC model, and we believe that the augmented model resolves three of these to varying extents: 1) an inaccurate match

to patient error patterns, 2) an incomplete account for semantic errors, and 3) no account for double letters.

#### 6.5.2.1 Inaccurate Match to Patient Data

In our augmented version of the model we examined in some detail how the model might produce different errors contingent on lesion location, lesion type, serial context type, and modification of thresholds. Each of these provided very different types of errors irrespective of which network was lesioned. In other words, it was possible to produce *type-B* symptoms by lesioning in network A, and *type-A* symptoms by lesioning in network B (see sections 6.4.1 and 6.4.2). In particular, applying the lesion type that Glasspool et al originally applied to network B (scale), and a qualitatively similar one (constrain) both to network A, seemed in many cases to produce *type-B* behaviour (see Table 6.6and Table 6.7). One might therefore argue that in some cases, it was lesion type and not the locus of impairment that dictated error behaviour. In any case, it was shown in Figure 6.9 that raising and lowering the letter threshold could provide a reasonable qualitative match to five GBD patients. It is likely that combining threshold modification with additional lesions could provide a match to other patients. We would claim that the fact that our results are based on the average of fifteen quasi patients across ten lesion severities shows that the error patterns are generally consistent in the presence of damage, and this is not something that Glasspool et al could lay claim to with their original testing approach. Specifically, a clear problem with their semantic design for network B is that it exposed the model to an inconsistent ability to produce more errors on abstract than concrete words.

### 6.5.2.2 Incomplete Account for Semantic Errors

We have already described how the semantic design for network B restricts the type of errors possible in the presence of damage. The design is structured to distinguish between abstract and concrete words, and includes no semantic information that may produce conceptually similar word as a consequence of error. We investigated use of a different semantic design, which seemed to produce semantic errors but as this was beyond the scope of the thesis, we did not pursue this in extensive detail. We describe an alternative design, which we believe may produce both a concreteness effect and semantic errors in Chapter 8.

### 6.5.2.3 No Account for Double Letters

The GSC model evolved from prior investigations into GBD (e.g. Houghton et al, 1994, Shallice et al, 1995, Glasspool, 1998, Glasspool & Houghton, 2005) and each model examined the effectiveness of a separate geminate representation in order to manage double letters. As discussed in Chapter 5, we provide a means of catering for double letters, but due to length of this chapter, we have deferred a fuller examination of this to Chapter 7. In summary however, we believe that the model provides a reasonable approximation to the types of errors associated with geminates shown by patients.

### 6.5.2.4 Summary

In summary, the GSC model appears to provide most of the underlying mechanisms required to produce symptoms of GBD. There are four reasons why we believe this is a good foundation for further development of models of graphemic buffer disorder. Firstly, the mechanisms seem robust in the presence of damage. We believe this has been validated by our use of quasi-patients and multiple lesion severities. Secondly the model seems to reflect symptoms associated with orthographic interrelationships which

were not predicted by the original model, namely, conformance to a minimum complexity principle, and production of fewer errors for simple CV words. Thirdly, each network can be considered separate functional entities in the sense that one might consider for example replacing network B at a future date with something more plausible. We have already expressed our reservations about the design of network B, but in defence of the overall architecture of the model, there are no constraints to how other networks may be added to the GOB (network A) in future. Lastly, we found it extremely simple to modify the augmented model to use multiple context types, which allowed us to make comparisons between them. As we discuss in the next chapter, adding a doubling facility was also fairly straight forward.

The following chapter completes the analysis of results associated with the augmented model by describing our investigations into approaches that may account for errors associated with geminates shown by patients.

# 7. Geminates

## 7.1 Introduction

Up to this point in the thesis, we have alluded to the ability of our model to cater for doubling (or geminate) errors. Other models based on competitive queuing principles have also attempted to explain geminate production (e.g. Houghton et al, 1994, Glasspool & Houghton (2005). One omission of the GSC model however, was the lack of a geminate mechanism, and in this chapter we investigate a number of approaches to resolving this and describe our results in relation to patient data.

There is strong empirical support for distinct and dissociable representations of letter identity and letter doubling information (e.g. Caramazza & Miceli, 1990, Tainturier & Caramazza, 1996, Miceli, Benvegnù, Capasso, & Caramazza, 1995, McCloskey et al, 1994). The cases in these papers describe some common behaviour associated with letter doubling, yet they do not seem consistent. For example, SFI (Miceli et al, 1995) had a selective deficit in the production of double letters leading to errors like *leggo*→LEGO, whereas the case of FM (Tainturier & Caramazza, 1996) showed the reverse pattern, with double information being much better preserved than letter identity and order (e.g. *umbrella*→UMMOUCAN, *ribbon*→BROLLOW). These types of behaviour suggest firstly, that double-letter sequences behave as units and secondly that information about grapheme quantity is encoded separately from information about grapheme identity. One case suggesting a separate geminate status concerns OM (Miozzo and De Bastiani, 2002). Of all inserts made by OM, 88.1% of these suggested a geminate addition (e.g. POLIZIA→POLIZZIA), and 46.7% of all deletes suggested a lost geminate (e.g. BOCCA→BOCA). OM's substitution behaviour also suggested a separate geminate status. Further, for all substitutions involving

geminates, 68 geminate errors (from 2401 errors in total) affected both letters (e.g. CANNOLI→CAMMOLI), whereas substitutions affecting only one letter (e.g. CANNA→CANMA) were recorded only 11 times. FM (Tainturier & Caramazza, 1996) showed more geminate substitutions (38.3%), and geminate movements (36.2%) than geminate insertions (4.3%) and AS (Jonsdottir et al, 1996) showed no geminate substitutions at all.

In this chapter we summarise our investigations into a geminate production mechanism based on the assumption that geminate status is stored separately from letter identity. Using this separate *dimension* as a core architectural feature of our network, we then proceed with building five models of geminate production, each adding a feature or features in an attempt to understand possible mechanisms behind geminate production. The aim of this exercise is to investigate those features important in providing robust geminate behaviour that can be shown to augment the GSC model. We also introduce eight classifications of geminate behaviour applicable to models of letter production and where appropriate, compare our results to available patient data.

## 7.2 Geminate Identity as a Separate Dimension

There are cases that suggest consonant/vowel status is represented separately within the language production system (e.g. Caramazza & Miceli, 1990, Miceli, Capasso, Benvegnù & Caramazza, 2004, McCloskey et al, 1994). The literature on geminate production is sparse however, and comes from a small number of cases. Caramazza & Miceli (1990) also proposed that geminates have a distinct identity within the language production system. They argue that orthographic representations consist of at least a consonant/vowel status *tier* and a grapheme identity tier (see also McCloskey et al, 1994 for their *Multiple Tier Hypothesis*). A graphical representation of these tiers can be seen

in Figure 3.10 (a) for the word BASKET, and in Figure 3.10 (b) for the word RABBIT, which shows the proposed doubling feature.


**Figure 7.1: (a) Representation of grapheme identity and consonant/vowel status, and (b) Representation of a doubling status as per Caramazza & Miceli (1990)**

Before Caramazza & Miceli (1990), Rumelhart & Norman (1982) described the phenomenon of geminate shifts in typing. For example, *look* can sometimes become *lokk*. They argue that such errors have two implications. Firstly, that there exist special *schemata* (or rules) signalling the existence of doubled letters, which are sometimes applied to the wrong letters. Secondly they claim "*the need for a special schema to mark double letters implies a difficulty in having the regular schema signal the double*". In other words, the word 'book' would not be represented by the schemata 'b', 'o', 'o', 'k' as this would require two tokens or instances of the schema for 'o', and the types of errors produced suggest that this is not the case, and that errors seem to suggest the existence of a distinct double *status*. Furthermore, in the absence of such a status, there would be more transpositions of constituent schemata leading to errors such as 'book→boko'. Such cases do exist, but it is thought that they arise from multiple errors rather than a simple transposition of one of two individual letters from a pair. A restriction of repeated schema instances also seems to accord with the notion of refractory competition (and inhibition) discussed in Chapter 5. Refractory competition among letters means that it is not possible to produce a letter twice in a row, and this is a core architectural feature of CQ models. Patient AS provides a notable, if uncommon counter example in spelling the word CANINE as CANNIE. A Rumelhart & Norman

interpretation of this error might suggest that a geminate was added to the first 'N' followed by a delete of the second 'N'. An alternate interpretation may be that the two tokens for the letter 'N' and 'I' in positions four and five have simply transposed. The latter interpretation is not possible with the GSC model as the inhibition mechanism prevents production of a letter immediately following another with the same identity, thus preventing the second 'N' from being a candidate for production in that position. The GSC model therefore behaves more in line with Rumelhart and Norman's interpretation of the error. Later in the chapter we describe how doubling errors such as CANINE→CANNIE may seem plausible in patient data despite a functional inhibitory mechanism.

### 7.2.1 Behaviour of Geminate Errors

In order to arrive at a working model of geminate production, we first describe the known behaviour of geminates. The following four behaviours are found in the literature as being typical doubling errors. These are not all applicable to every patient showing a geminate related disorder. However, in order to demonstrate patient-like behaviour, we assume that a working model should be able to demonstrate as many of these symptoms as possible:

1) While letter identity may be impaired, doubling information is often preserved. This may lead to another letter being doubled in place of the original. For example, PARROT → PATTOR by patient FM. Note also the transposition.
2) The geminate status is not *hard-wired* to the position in the word being doubled. So the double may appear elsewhere in the word, for example, BREEZE → BREZZE by patient AS.
3) A geminate status can be lost. E.g. BOCCA → BOCA by patient OM. Badecker (1996) has termed this *geminate shortening*.

4) Words containing no doubles rarely show geminate additions. For example, in delayed copy transcoding tests, FM only generated additions in words already containing double letters. This might suggest that while geminate existence relates to letter position, it might also be held at the level of the entire word. There are however counter examples cases. For example, in other tests, FM produced geminates in words without existing double letters. The authors claim that this may be due to a two-step process involving first a semantic error followed by a graphemic buffer error on the new word. One instance discussed by Tainturier & Caramazza (1996) is the response ELEPHANT → ROTIGGE, which they argue could arise as follows: ELEPHANT → GIRAFFE → ROTIGGE. Note the consistency in geminate position and adherence to consonant/vowel status. Another notable exception to this behaviour was shown by AS, who produced almost 50% of geminate related errors on words without doubles. We investigate this specific behaviour later in the chapter

### 7.2.2 Description of possible geminate activity

One advantage of a model is that we can view all steps involved in the production process, and it is clear whether a letter has been omitted rather than perhaps switched or exchanged. It is also clear whether doubling is due to an active geminate status, or some other process. We now describe eight classifications of geminate error that may be produced by our models. Each classification references a fictional spelling error to demonstrate the internal processing of the model, and uses the notation introduced in Chapter 5 to describe these behaviours. For example, a letter with an accompanying asterisk (e.g. **E***) describes a letter with an active geminate status, and a letter in square brackets (e.g. **[E]**) shows that the winning letter did not reach presentation threshold and was deleted.

1) *Geminate Movement*

This is where a word's geminate status moves to another position, and termed *Geminate Shift* by McCloskey et al (1994). By definition, this requires the loss of a geminate status somewhere in the word, and a corresponding geminate added to different position. Examples of this include KNEE → KN*E → KNNE, or SUPPOSE → SUPOS*E → SUPOSSE.

2) *Letter Substitution*

This is where a word's geminate status does not move, yet the letter being doubled changes. Examples of this include KNEE → KNO* → KNOO, or SUPPOSE → SUB*OSE → SUBBOSE.

3) *Lost Status*

This is where a word containing a double is recalled with no active geminate status. Examples of this include KNEE → KNE, or SUPPOSE → SUPOSE.

4) *Additions with no source geminate*

This is where a word containing no doubles is recalled with a letter having an active geminate status. An examples of this is IRIS → IRI*S → IRIIS.

5) *Additions with source geminate*

This is where a word containing doubles has additional letters with active geminate statuses in the recalled word. Examples of this include, SPOON → SPO*N* → SPOONN.

All five behaviours described above have been seen in patient data (see Table 7.2). Although McCloskey et al (1994), did not define a category for geminate additions for their patient HE despite providing what we believe is at least one plausible example (MAGAZINE → MAGGAIZE).

We now describe three behaviours, which can be seen in our model of spelling due

to the transparency of the processing mechanism. We are aware, for example, when a letter has been deleted, which is not always obvious with patients. Therefore, while the following three classifications *may* occur with patients, we are not in a position to substantiate them.

6) *Pseudo additions with source geminate*

This is where a word containing doubles has double letters in the recalled word that do not occur as a result of an active geminate status. Examples of this include, CHEERIER → CHE*R[I][E]R → CHEERR.

7) *Pseudo additions with no source geminate*

This is where a word without doubles has double letters in the recalled word that do not occur as a result of an active geminate status. Examples of this include, IRIS → I[R]IS → IIS.

8) *Pseudo geminate movement*

This is where a word containing doubles loses a geminate status at one position, yet creates another geminate via a pseudo addition. An examples of this is CHEERIER → CHERI[E]IR → CHERIIR.

As we will now show, the last three classifications predict that triples may also be possible. If we assume that in an intact system, the letter production mechanism is constrained to producing single letters due to an inhibition process. At most, one should see doubles due to an active geminate status, albeit substituted, transposed, or added. However, triples have been seen in patients. For example AS showed two triples, SLEEVE → SLEEEVE, and MAROON→ MOOON. One possible explanation for triples is a strong reduction or deficiency in $GABA_A$ receptor-mediated inhibition, which could result in *runaway* excitation. In the model this might be explained by reducing the refractory or inhibitory effect on letters immediately following their

presentation, allowing them to reappear more easily. A second explanation is that the *doubling* mechanism is faulty allowing it to generate triples. An alternative explanation for triples is that *invisible* deletes actually do occur, thus satisfying the refractory process, yet producing excessive numbers of the same letter. One example of this may be interpreted in results from patient AS who recalled the word BANANA as BANNA. If a letter is immediately inhibited following its production to prevent another separate occurrence of that letter, then we might view AS's response in two ways. One view is that a geminate was added to the first 'N' followed by a deletion of the following 'A', and 'N'. An alternative and considerably simpler view is that the letter 'A' in position four was simply deleted. This may be interpreted in at least three other examples by AS. The first is BIKINI → BIIM, with a deleted 'K'. The second is SLEEVE → SLEEEVE, with possibly a deleted 'V' after the second 'E', followed by two within-word perseverative additions. The third is MAROON → MOOON, with the first 'A' substituted by a within-word vowel, followed by an omission of the letter 'R'.

If we cannot prove that pseudo geminates actually do exist in patients, we have two choices. Firstly, we can ignore them, as they are not *real* doubles but artefacts of interspersed deletes rather than the result of an active geminate status. Alternatively, we cannot prove that this behaviour does not occur in patients, and may therefore be entirely plausible. In presenting our results, we will show all classifications. While we can make no claim for either case, it is of interest to show both views of the data, allowing the reader to make their own judgement.

### 7.2.3 Comparison with Empirical Data

Caramazza & Miceli (1990) describe a number of geminate related classifications with their Italian patient LB and we provide a mapping to our classifications in Table 7.1, to show that we can accommodate their classifications. Caramazza & Micelli differentiate

between various geminate-movement types in accord with their transposition classifications, namely geminate *exchanges* and *shifts*. We combine both movement types into a single classification as we have done so far in the thesis relating to the transpositions of single letters. Further, Caramazza & Micelli only discuss geminated consonants, which are consistent with the relative rarity of geminated vowels in Italian (Miozzo & Bastiani, 2002). For the purpose of the thesis, we do not differentiate between geminated consonants and vowels. Indeed, Jonsdottir et al (1996) performed a comparative analysis of this on the English patient AS, finding no noticeable difference between the performance of geminated consonants and vowels.

| C&M Error Classification | Example | Geminate Classification |
|---|---|---|
| Substitution of the geminate consonants | SORELLA → SORE**TT**A | Letter Substitution |
| Geminate duplication | SORELLA → SO**RR**ELLA | Additions with source geminate |
| Deletion of a geminate consonant | SORELLA → SORE**L**A | Lost Geminate |
| Exchange involving the geminate consonant | SORELLA → SOLE**RR**A | Letter Substitution (*and transposition*) |
| Shift of the geminate feature | SORELLA → SO**RR**ELA | Geminate Movement |
| Exchange of the geminate feature and consonant | SORELLA → SO**LL**ERA | Geminate Movement (and letter transposition) |
| Substitution of one geminate consonant | SORELLA → SORE**LT**A | Lost Geminate (and inserted character) |

**Table 7.1 : Mapping Caramazza & Micelli geminate behaviour to our classifications**

### 7.2.4 Patent Data being investigated

Our core comparative data (Table 7.2) comes from three patients, Two English speaking patients AS (Jonsdottir et al 1996), and FM (Tainturier & Caramazza, 1996), and an Italian-speaking patient LB (Caramazza & Miceli, 1990). Patient AS was diagnosed with normal pressure hydrocephalus arising as a later development of a head injury requiring hospitalisation sustained 20 years earlier, and there was no evidence of an infarct. We would categorise the nature of hydrocephalus as being diffuse in nature, although some focal damage would be expected to occur as a direct result of the brain

being pressed against the skull. Patient LB suffered from a cerebrovascular accident involving the parietal areas of the left hemisphere. A computed tomography scan showed a hypodense area involving the superficial and deep parietal structures of the left hemisphere, which we believe would suggest focal lesioning. Patient FM suffered a large infarct of the left middle cerebral artery. A CT scan performed two years later revealed a large area of damage involving the infero-posterior frontal lobe, the inferior parietal lobe, and the anterior temporal lobe of the left hemisphere, as well as the underlying white matter and lateral basal ganglia; there was also evidence of cortical atrophy of the remainder of the left frontal convexity. Despite the large area involved in the infarct, we would also categorise the nature of the FM's lesion as focal.

| Error Type | AS | LB | FM |
|---|---|---|---|
| Geminate Movement | 19.1% | 52.1% | 36.2% |
| Letter Substitution in geminates | 0.0% | 8.7% | 38.3% |
| Lost Status | 28.6% | 20.2% | 21.3% |
| Additions (no source geminate) | 46.7% | 0.2% | 0.0% |
| Additions (source geminate) | 5.7% | 18.8% | 4.26% |

**Table 7.2 : A summary of the geminate error behaviour of a number of patients.**

We chose the above patients for a number of reasons. Firstly, they provide results across two languages, suggesting that geminate behaviour is broadly similar across languages, which can be seen in the qualitative similarities between LB and FM. Secondly, although authors have presented interpretations of some geminate related behaviour for other patients, much of this has not been collated in sufficient detail to make a comprehensive enough analysis for our purposes. Thirdly, and most importantly, at least one patient AS shows some apparently contradictory results to LB and FM. We feel that this highlights a greater complexity in the geminate production mechanism, that would not have been as obvious were all patient data quite qualitatively similar. It is possible that the patient aetiology was in some way responsible for the nature of their results, and we will investigate different lesioning approaches in more detail later in the chapter.

A number of other points should be mentioned regarding the data in Table 7.2. Results for AS were analysed by us from the raw data. In Jonsdottir et al's original analysis, the authors omitted geminate additions for words not possessing source geminates. Therefore, their category *geminate duplication* (e.g. pepper → peepper) did not properly reflect the number of geminate additions produced by AS. Our analysis of their results therefore shows relatively fewer geminate movements and lost status values than their original published data. For LB's data, there appears to be an error in the published result by Caramazza & Miceli (1990). The authors assign 27.5% of errors to exchanges involving the geminate consonant. This occurred 10 times from 69 total geminate related errors, which should have been given the value 14.5%. We also do not discriminate between different geminate movement types. Where the authors reference geminate exchanges (14.5%), geminate shifts (36.2%), and exchanges of geminate feature and consonant (1.4%), we collate these into a single geminate movement classification having the value 52.1%. Our value for lost geminate status is calculated as the sum of two error types described by Caramazza & Miceli. The first is geminate deletions such as sorella→sorela (13%). The second is the *implicit* delete resulting from one of a pair of consonants being substituted such as sorella->sorelta (7.2%). In keeping with our interpretation of geminate behaviour, we consider this a lost geminate and a character addition. FM's results are based on our analysis of the raw data of delayed copy transcoding errors.[23].

In comparing patients results, the anomalous behaviour seems to belong to AS for at least two reasons. Firstly, FM and LB show different levels of letter substitution, yet AS shows no geminate substitution errors at all. Although another patient SFI (Miceli et al, 1995) showed no substitutions, there was little other data available to make

[23] Provided by Marie-Josephe Tainturier

a fair comparison to our three patients above. Secondly, both FM and LB show relatively few additions in words not containing doubles, yet show evidence of additions in words already containing doubles, and AS shows the reverse pattern. All show similar percentages of geminate deletes, and varying degrees of geminate movement.

### 7.2.4.1 Simulation Options

The foundation for our geminate production model is Network A described in Chapter 5. However, the GSC corpus contains very few geminates, so we created a new corpus containing 200 words (see Appendix C) based on the 1000 word corpus of Ward (1997). Our reason for using a smaller corpus is primarily motivated by the practicality of time taken to run simulations. We retain the overall nature of Ward's corpus by ensuring a roughly comparable distribution of grapheme identity, word length, and geminate count. Table 7.3 lists the word length and geminate frequencies for each corpus. In order to provide similar word lengths to corpora used elsewhere in the thesis, we only used words containing four to seven letters. With a smaller corpus size, we also scaled down the number of hidden units to 75, retaining the same proportion of hidden units to corpus entries as that used by Glasspool et al (400 corpus items and 150 hidden units).

| Word Length | Ward (1997) | | New Corpus | |
|---|---|---|---|---|
| | Words (%) | With Geminates (%) | Words (%) | With Geminates (%) |
| 3 | 7.30 | 0.40 | - | - |
| 4 | 26.90 | 3.40 | 30.50 | 4.50 |
| 5 | 30.40 | 4.70 | 34.50 | 5.00 |
| 6 | 20.50 | 4.90 | 23.50 | 7.00 |
| 7 | 9.90 | 1.60 | 11.50 | 3.00 |
| 8 | 5.00 | 0.30 | - | - |

**Table 7.3 : Word Length and Geminate Distribution of the corpora used by Ward (1997) and in this study.**

Table 7.3 presents a word and geminate frequency analysis of both corpora where there seems to be a qualitatively similar distribution across word length, and geminates by word length. Table 7.4 presents a lexical analysis of both corpora showing their relative

letter frequencies. It should also be noted that since we limited the new corpus to valid English words, it was difficult to get an exact match of letter distribution.

| Letter | Ward (1997) % | New Corpus % | Letter | Ward (1997) % | New Corpus % |
|---|---|---|---|---|---|
| A | 8.564 | 8.624 | N | 5.742 | 5.717 |
| B | 2.783 | 2.810 | O | 7.084 | 7.558 |
| C | 4.807 | 4.845 | P | 3.192 | 2.616 |
| D | 3.640 | 3.488 | Q | 0.195 | 0.194 |
| E | 12.417 | 12.791 | R | 6.948 | 7.655 |
| F | 1.752 | 1.744 | S | 5.644 | 6.008 |
| G | 2.569 | 2.422 | T | 6.442 | 6.492 |
| H | 3.075 | 3.004 | U | 3.912 | 2.713 |
| I | 5.625 | 5.523 | V | 1.109 | 1.163 |
| J | 0.448 | 0.484 | W | 1.440 | 1.357 |
| K | 1.557 | 1.550 | X | 0.195 | 0.194 |
| L | 5.722 | 5.717 | Y | 1.927 | 1.938 |
| M | 2.919 | 2.907 | Z | 0.292 | 0.484 |

**Table 7.4 : Lexical analyses of the corpora used by Ward (1997) compared to the new corpus.**

Table 7.5 describes the distribution of geminate positions for each corpus. The scoring for this was obtained by allocating a point to each letter position containing a double letter. The word BULLET would therefore have a point allocated to positions three and four. In Ward's corpus, geminates positions for each word length seem to show a roughly normal distribution with more geminates located towards the centre of words. Our corpus showed similar characteristics.

| Letter | Word Length - Ward (1997) | | | | | | Word Length – New Corpus | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Position | 3 | 4 | 5 | 6 | 7 | 8 | 4 | 5 | 6 | 7 |
| 1 | 0.0% | 0.0% | 0.0% | 0.0% | 0.0% | 0.0% | 0.00% | 0.00% | 0.00% | 0.00% |
| 2 | 50.0% | 25.0% | 11.7% | 4.1% | 6.3% | 0.0% | 22.2% | 15.0% | 0.00% | 8.3% |
| 3 | 50.0% | 50.0% | 37.2% | 39.2% | 34.4% | 0.0% | 50.0% | 30.0% | 39.3% | 33.3% |
| 4 | - | 25.0% | 38.3% | 42.3% | 37.5% | 33.3% | 27.8% | 35.0% | 50.0% | 25.0% |
| 5 | - | - | 12.8% | 10.3% | 12.5% | 50.0% | - | 20.0% | 10.7% | 16.7% |
| 6 | - | - | - | 4.1% | 6.3% | 16.7% | - | - | 0.00% | 16.7% |
| 7 | - | - | - | - | 3.1% | 0.0% | - | - | - | 0.00% |
| 8 | - | - | - | - | - | 0.0% | - | - | - | - |

**Table 7.5 : Distribution of Geminate positions within each corpus.**

## 7.3 Error distribution

To clarify whether a new corpus would change the error distribution, and thus influence overall results, we carried out an examination on the relative error types for both corpora using a wide variety of lesion types and locations (Table 7.6).

| Lesion Location | Lesion Type | Glasspool Corpus % | | | | New Corpus % | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Subs | Ins | Txps | Dels | Subs | Ins | Txps | Dels |
| Random | Ablate | 63.32 | 0.86 | 17.29 | 18.53 | 67.37 | 0.87 | 15.98 | 15.78 |
| | Noise | 66.27 | 0.51 | 17.60 | 15.62 | 67.16 | 0.34 | 20.00 | 12.49 |
| | Constrain | 35.80 | 0.87 | 5.68 | 57.66 | 39.20 | 0.94 | 9.72 | 50.14 |
| | Scale | 40.49 | 0.66 | 2.54 | 56.31 | 31.13 | 1.68 | 12.16 | 55.03 |
| Input | Ablate | 61.41 | 0.85 | 18.66 | 19.08 | 64.78 | 0.64 | 15.49 | 19.09 |
| | Noise | 66.03 | 0.49 | 16.62 | 16.87 | 64.84 | 0.47 | 19.11 | 15.57 |
| | Constrain | 55.30 | 0.52 | 10.67 | 33.52 | 50.14 | 0.87 | 15.95 | 33.03 |
| | Scale | 54.04 | 0.63 | 8.01 | 37.31 | 48.05 | 1.07 | 19.38 | 31.51 |
| Input Context | Ablate | 59.49 | 0.98 | 18.26 | 21.26 | 62.87 | 0.72 | 16.81 | 15.59 |
| | Noise | 66.81 | 0.54 | 14.28 | 18.37 | 64.66 | 0.34 | 18.73 | 16.27 |
| | Constrain | 63.59 | 1.02 | 13.70 | 21.70 | 55.88 | 0.54 | 17.09 | 26.49 |
| | Scale | 63.35 | 1.19 | 12.00 | 23.46 | 55.05 | 0.45 | 18.38 | 26.12 |
| Input Word Identity | Ablate | 59.05 | 0.89 | 15.87 | 24.20 | 62.65 | 0.54 | 15.88 | 20.92 |
| | Noise | 65.41 | 0.84 | 16.36 | 17.40 | 65.42 | 0.51 | 17.09 | 16.99 |
| | Constrain | 43.92 | 0.29 | 5.93 | 49.86 | 46.27 | 0.81 | 18.75 | 34.17 |
| | Scale | 42.44 | 0.29 | 5.52 | 51.75 | 45.03 | 1.15 | 20.08 | 33.73 |
| Hidden | Ablate | 63.10 | 0.88 | 17.12 | 18.90 | 68.54 | 0.77 | 15.44 | 15.25 |
| | Noise | 66.99 | 0.51 | 16.94 | 15.56 | 69.87 | 0.44 | 17.41 | 12.28 |
| | Constrain | 42.76 | 0.61 | 5.38 | 51.26 | 41.39 | 0.79 | 13.94 | 43.88 |
| | Scale | 1.04 | 1.51 | 0.00 | 97.45 | 0.04 | 0.31 | 0.00 | 79.66 |
| Output | Noise | 62.98 | 0.45 | 18.81 | 17.77 | 63.61 | 0.45 | 18.43 | 17.51 |

**Table 7.6: Error distributions for total errors for the original Glasspool as well as the new corpus.**

This examination covers all errors including those relating to single letters. If this shows a qualitatively similar performance between corpora, we can assume that geminate related tests on a larger corpus would tend to behave in a similar fashion to the way they do in our smaller corpus. As with our tests in Chapter 6, our results are calculated by averaging the errors from fifteen *quasi-subjects* across ten progressively increasing lesion severities. Table 7.7 shows descriptive statistics for the error values produced by each corpus showing that they indeed seem to be qualitatively similar and we therefore believe that the new corpus provides a comparable basis for our experiments.

| | Glasspool Corpus | | | | New Corpus | | | |
|---|---|---|---|---|---|---|---|---|
| | Subs | Ins | Txps | Dels | Subs | Ins | Txps | Dels |
| Mean | 54.46 | 0.73 | 12.24 | 32.56 | 54.00 | 0.70 | 16.00 | 28.17 |
| Std Dev. | 15.77 | 0.30 | 5.98 | 21.07 | 16.65 | 0.33 | 4.47 | 17.12 |

**Table 7.7: Error distributions for total errors for the original Glasspool as well as the new corpus.**

## 7.4 Towards a model of Geminate Production

To investigate aspects of an eventual geminate production mechanism, which may be susceptible to the same error behaviour in the presence of lesioning as patients, we created five different models (A → E) of the graphemic buffer network shown in Figure 7.2, each progressively improving upon the previous. Each model is trained using a momentum of 0.9, and a learning rate of 0.001.

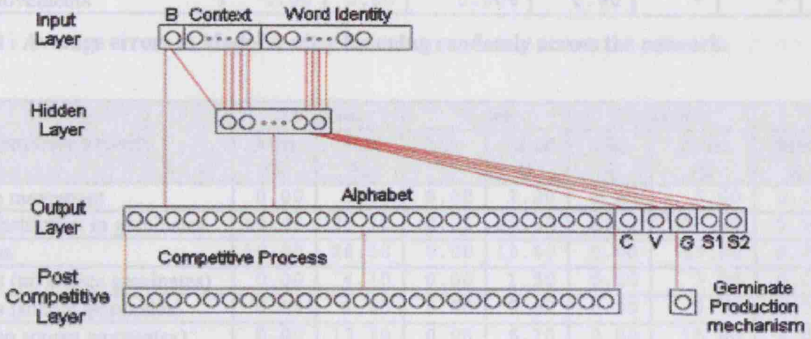



**Figure 7.2 : The Graphemic Output Buffer network**

We use three random seeds for training and five random seeds for recall on each, simulating fifteen subjects per model. A geminate status is deemed active if its activation exceeds a given threshold, similar to the approach used for standard letter presentation. For each model, we apply ablation, noise, constraint, and scaling randomly across the network to simulate diffuse lesioning. Later in the chapter, we apply ablation and noise in a focal manner to provide a comparative analysis. We now discuss each model and its performance providing two tables of results for each. The first shows the average number of errors across all lesion severities and the second shows minimum and maximum error rates for each lesion type.

### 7.4.1 Model A

In our first model, the geminate status is implemented using a simple ON/OFF flag. Where a letter requires doubling, the desired geminate status is set to 1.0 for ON,

otherwise it is set to 0.0 for OFF. If the geminate value exceeds a threshold on recall, it is considered active and the *winning* letter is output twice.

| Geminate activity | Ablation % | Noise % | Constrain % | Scale % | AS % | LB % | FM % |
|---|---|---|---|---|---|---|---|
| Geminate movement | 0.30 | 0.67 | 0.60% | 0.00 | 19.1 | 52.1 | 36.2 |
| Letter Substitution in geminates | 71.43 | 88.11 | 53.35% | 56.13 | 0.0 | 8.7 | 38.3 |
| Lost status | 19.68 | 8.56 | 35.94% | 25.50 | 28.6 | 20.2 | 21.3 |
| Additions (no source geminates) | 1.20 | 0.22 | 1.30% | 1.75 | 46.7 | 0.2 | 0.0 |
| Additions (source geminates) | 0.40 | 0.22 | 0.00% | 0.00 | 5.7 | 18.8 | 4.26 |
| Pseudo (no source geminates) | 6.19 | 2.22 | 8.81% | 16.63 | - | - | - |
| Pseudo (source geminates) | 0.80 | 0.00 | 0.00% | 0.00 | - | - | - |
| Pseudo movements | 0.00 | 0.00 | 0.00% | 0.00 | - | - | - |

**Table 7.8 : Average errors in Model A after lesioning randomly across the network.**

| Geminate activity | Ablation | | Noise | | Constrain | | Scale | |
|---|---|---|---|---|---|---|---|---|
| | Min % | Max % | Min % | Max % | Min % | Max % | Min % | Max % |
| Geminate movement | 0.00 | 1.00 | 0.00 | 2.20 | 0.00 | 3.00 | 0.00 | 0.00 |
| Letter Substitution in geminates | 60.40 | 89.70 | 0.00 | 95.50 | 21.2 | 100.00 | 0.00 | 100.00 |
| Lost status | 10.30 | 28.50 | 0.00 | 13.60 | 0.00 | 63.60 | 0.00 | 69.40 |
| Additions (no source geminates) | 0.00 | 4.10 | 0.00 | 1.50 | 0.00 | 3.00 | 0.00 | 7.10 |
| Additions (source geminates) | 0.00 | 1.60 | 0.00 | 0.80 | 0.00 | 0.00 | 0.00 | 0.00 |
| Pseudo (no source geminates) | 0.00 | 13.20 | 0.00 | 6.10 | 0.00 | 15.80 | 0.00 | 50.00 |
| Pseudo (source geminates) | 0.00 | 2.40 | 0.00 | 0.40 | 0.00 | 0.00 | 0.00 | 0.00 |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |

**Table 7.9 : Minimum & Maximum percentages of Model A after lesioning.**

Comparing the performance of Model A to patient results, a simple ON/OFF approach seems problematic. Ignoring Pseudo additions, Table 7.8 shows few geminate additions, irrespective of whether there were geminates in the source word. This also suggests that a target value of 0.0 may be excessive for letters not requiring doubling, as it may be more difficult for them to be incorrectly doubled in the presence of lesioning. We also observed that the constraint and scaling lesion types were more prone to pseudo additions due to interspersed letter deletions. For example, NEPHEW → NE[**P**][**H**]E[**W**]→NEE. This is expected as the constraint and scaling lesion types reduce overall activation fed forward to the competitive filter.

In comparing the performance of model A to the published data, the model appears unable to adequately explain patient results. One exception is the loss of

geminate status as a result of ablation, which shows similar results to the percentage of those produced by patients. Another exception is in the general tendency of almost all lesion types to produce more additions in words without source geminates than in words with geminates. This pattern is also present in AS.

### 7.4.2 Model B

The first modification to our model introduces a lazy updating approach for learning the geminate status (Glasspool, 1998). The network is trained to recognise geminate existence and ignores the activation of non-geminated letters allowing them to retain any value below the activation threshold.

| Geminate activity | Ablation % | Noise % | Constrain % | Scale % | AS % | LB % | FM % |
|---|---|---|---|---|---|---|---|
| Geminate movement | 0.60 | 0.50 | 2.70 | 2.30 | 19.1 | 52.1 | 36.2 |
| Letter Substitution in geminates | 37.46 | 21.00 | 39.56 | 25.40 | 0.0 | 8.7 | 38.3 |
| Lost status | 14.49 | 3.20 | 42.56 | 42.90 | 28.6 | 20.2 | 21.3 |
| Additions (no source geminates) | 28.47 | 43.30 | 0.20 | 0.00 | 46.7 | 0.2 | 0.0 |
| Additions (source geminates) | 15.19 | 30.80 | 0.20 | 0.40 | 5.7 | 18.8 | 4.26 |
| Pseudo (no source geminates) | 3.50 | 1.10 | 14.79 | 29.00 | - | - | - |
| Pseudo (source geminates) | 0.30 | 0.10 | 0.00 | 0.00 | - | - | - |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | - | - | - |

**Table 7.10 : Average errors in Model B after lesioning randomly across the network.**

| Geminate activity | Ablation | | Noise | | Constrain | | Scale | |
|---|---|---|---|---|---|---|---|---|
| | Min % | Max % | Min % | Max % | Min % | Max % | Min % | Max % |
| Geminate movement | 0.00 | 1.30 | 0.00 | 1.30 | 0.00 | 6.70 | 0.00 | 9.10 |
| Letter Substitution in geminates | 19.80 | 94.10 | 14.10 | 33.30 | 13.90 | 63.00 | 0.00 | 50.00 |
| Lost status | 0.90 | 29.20 | 0.60 | 8.50 | 16.70 | 70.80 | 0.00 | 72.70 |
| Additions (no source geminates) | 0.00 | 39.90 | 16.70 | 51.70 | 0.00 | 0.90 | 0.00 | 0.00 |
| Additions (source geminates) | 0.00 | 30.80 | 22.40 | 44.40 | 0.00 | 1.00 | 0.00 | 4.00 |
| Pseudo (no source geminates) | 0.00 | 6.40 | 0.00 | 3.10 | 6.90 | 25.00 | 11.80 | 100.00 |
| Pseudo (source geminates) | 0.00 | 0.80 | 0.00 | 0.40 | 0.00 | 0.00 | 0.00 | 0.00 |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |

**Table 7.11 : Minimum & Maximum percentages of Model B after lesioning.**

This approach brings a number of benefits to our model. The first is that it seems more theoretically plausible than using ON/OFF targets as the model only learns to recognise active geminate statuses ignoring geminate absences. Secondly geminate activation for letters without a target active geminate status may be sufficiently close to the geminate

threshold to be activated in the presence of lesioning, thus allowing many letters to be doubled in error, a notable deficiency of model A. The third benefit is that a geminate status can be easily lost, as the activation only needs to drop below the threshold for this to occur. It is important to note that using lazy updating, geminate activation is not reinforced during training once it exceeds the required threshold. Activation levels will therefore tend to reside just above the geminate threshold facilitating easy loss during lesioning. This is apparent with constraint and scaling, but unfortunately, there is no selectivity in the propensity for geminates to appear. Despite the behaviour of patient AS, patients usually produce doubling errors in words with existing doubles, yet Table 7.10 shows that even words without source geminates are quite susceptible to letter doubling. The lazy updating mechanism only requires non-geminates to be below the presentation threshold to ensure they are not doubled. It does not prevent them from being very close to that threshold, so in the presence of lesioning, every letter is equally likely to have its geminate status driven above the threshold. This is obviously problematic for the model, as it does not reflect the general tendency of patients to make doubling errors in words with existing geminates. More geminate movement however was noted than with Model A, and this may be due to the fact that geminate additions and deletions were more likely to appear in this model. The presence of both in a single word will interpreted as a geminate movement.

The performance of model B relative to patients LB and FM is poor. Patient AS however, shows a relatively good fit to some of the data, as geminate additions in words with no existing doubles are produced more frequently than in words with existing doubles. This pattern was consistent with both the ablation and noise lesion types. The fact that few additions occurred with constrain and scaling lesion types may be due to an overall reduction in activation function values due to the nature of these lesion types –

they both reduce overall activity. Letter substitutions seem to be more consistent than deletions across lesion types, contrary to patient data which shows more consistency in deletions than substitutions. Despite the number of geminate additions being qualitatively similar to those produced by AS, geminate movement is still very low, highlighting a major inadequacy of Model B.

### 7.4.3 Model C

Our third model introduces a lower *non-geminate* threshold below which letters without a target geminate status are pushed during training. The motivation behind this threshold is to prevent an excess of letters being doubled that might otherwise sit just below the geminate activation threshold as exhibited in Model B.

| Geminate activity | Ablation % | Noise % | Constrain % | Scale % | AS % | LB % | FM % |
|---|---|---|---|---|---|---|---|
| Geminate movement | 0.70 | 0.70 | 0.70 | 0.00 | 19.1 | 52.1 | 36.2 |
| Letter Substitution in geminates | 57.50 | 82.62 | 15.10 | 6.69 | 0.0 | 8.7 | 38.3 |
| Lost status | 31.70 | 15.09 | 80.00 | 90.41 | 28.6 | 20.2 | 21.3 |
| Additions (no source geminates) | 2.70 | 0.20 | 0.10 | 0.00 | 46.7 | 0.2 | 0.0 |
| Additions (source geminates) | 2.90 | 0.10 | 0.70 | 1.20 | 5.7 | 18.8 | 4.26 |
| Pseudo (no source geminates) | 4.00 | 1.20 | 3.40 | 1.70 | - | - | - |
| Pseudo (source geminates) | 0.30 | 0.10 | 0.00 | 0.00 | - | - | - |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | - | - | - |

**Table 7.12 : Average errors in Model C after lesioning randomly across the network.**

| Geminate activity | Ablation | | Noise | | Constrain | | Scale | |
|---|---|---|---|---|---|---|---|---|
| | Min % | Max % | Min % | Max % | Min % | Max % | Min % | Max % |
| Geminate movement | 0.00 | 1.60 | 0.00 | 2.40 | 0.00 | 2.10 | 0.00 | 0.00 |
| Letter Substitution in geminates | 39.40 | 89.50 | 68.0 | 100.00 | 3.70 | 60.90 | 0.00 | 40.00 |
| Lost status | 7.90 | 51.10 | 0.00 | 28.70 | 39.10 | 90.80 | 60.00 | 100.00 |
| Additions (no source geminates) | 0.00 | 8.00 | 0.00 | 0.70 | 0.00 | 0.50 | 0.00 | 0.00 |
| Additions (source geminates) | 0.00 | 6.70 | 0.00 | 0.40 | 0.00 | 1.60 | 0.00 | 4.20 |
| Pseudo (no source geminates) | 0.00 | 8.80 | 0.00 | 4.70 | 0.00 | 7.00 | 0.00 | 7.30 |
| Pseudo (source geminates) | 0.00 | 1.00 | 0.00 | 0.70 | 0.00 | 0.00 | 0.00 | 0.00 |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |

**Table 7.13 : Minimum & Maximum percentages of Model C after lesioning.**

In Figure 7.3, this lower threshold is labelled $T_N$.

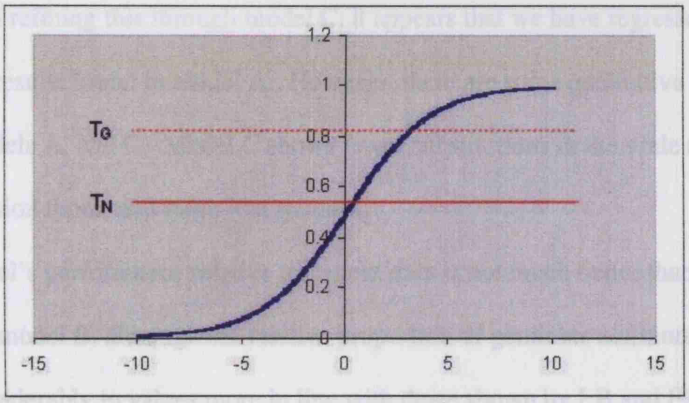



**Figure 7.3: Geminate and non-geminate status values as applied to a geminate's activation**

In Figure 7.3, $T_G$ is the status above which letters are activated for doubling, and $T_N$ is a lower threshold below which the activation of non-geminate letters must reside.

Training follows a lazy updating method, with four scenarios:

1. The geminate activation value exceeds a given geminate threshold $T_G$, and the letter **requires** doubling.
2. The geminate activation value exceeds a given non-geminate threshold $T_N$ and the letter **does not** require doubling.
3. The geminate activation value does not reach a given geminate threshold $T_G$, and the letter **requires** doubling.
4. The geminate activation value does not reach a given non-geminate threshold $T_N$ and the letter **does not** require doubling.

In scenarios 1 and 4, the geminate status is deemed correct, and no weight modifications are made. In scenario 2, we punish the geminate activation, and in scenario 3, we reinforce the desired geminate activation.

An initial glance at the results of models A, B, and C shows some interesting results. Firstly, model B promoted too many doubles due to the nature of the lazy updating

approach. In refining this through model C, it appears that we have regressed back to the types of results found in model A. However, there are some qualitative differences between models A, and C. Model C shows fewer substitutions in the scale and constraint lesion types, and more lost statuses.

The model's performance relative to patient data is not much better than that provided by model B, although the relative proportion of geminate additions has reduced considerably to values more in line with those shown by LB and FM. There does not however, seem to be any obvious bias of additions occurring in words with source geminates as shown by all three patients in one direction or the other. This is expanded on further in Model D.

### 7.4.4 Model D

This model assumes that geminate existence is known at the word level and may explain why words with existing geminates are more likely to have doubling errors than those without. This also predicts a differentiation mechanism between words with geminates and those without. It expands on model C by using different non-geminate thresholds in words with source geminates, such that constituent letters have a higher propensity for doubling.

| Geminate activity | Ablation % | Noise % | Constrain % | Scale % | AS % | LB % | FM % |
|---|---|---|---|---|---|---|---|
| Geminate movement | 0.40 | 0.10 | 0.50 | 0.00 | 19.1 | 52.1 | 36.2 |
| Letter Substitution in geminates | 56.50 | 66.63 | 14.50 | 12.20 | 0.0 | 8.7 | 38.3 |
| Lost status | 23.90 | 17.28 | 62.40 | 66.60 | 28.6 | 20.2 | 21.3 |
| Additions (no source geminates) | 1.60 | 0.20 | 0.20 | 0.10 | 46.7 | 0.2 | 0.0 |
| Additions (source geminates) | 13.20 | 13.39 | 5.00 | 10.00 | 5.7 | 18.8 | 4.26 |
| Pseudo (no source geminates) | 4.10 | 2.30 | 7.20 | 10.80 | - | - | - |
| Pseudo (source geminates) | 0.30 | 0.10 | 0.20 | 0.30 | - | - | - |
| Pseudo movement | 0.00 | 0.00 | 0.00 | 0.00 | - | - | - |

**Table 7.14 : Average errors in Model D after lesioning randomly across the network.**

| Geminate activity | Ablation | | Noise | | Constrain | | Scale | |
|---|---|---|---|---|---|---|---|---|
| | Min % | Max % | Min % | Max % | Min % | Max % | Min % | Max % |
| Geminate movement | 0.00 | 1.00 | 0.00 | 0.60 | 0.00 | 1.90 | 0.00 | 0.00 |
| Letter Substitution in geminates | 40.50 | 81.60 | 7.70 | 82.50 | 4.40 | 41.00 | 2.20 | 33.30 |
| Lost status | 5.30 | 41.10 | 7.20 | 30.90 | 33.30 | 85.70 | 0.00 | 88.90 |
| Additions (no source geminates) | 0.00 | 7.00 | 0.00 | 1.20 | 0.00 | 0.90 | 0.00 | 1.10 |
| Additions (source geminates) | 6.30 | 30.80 | 2.20 | 61.50 | 0.90 | 12.80 | 1.10 | 33.30 |
| Pseudo (no source geminates) | 0.00 | 7.20 | 0.00 | 5.80 | 4.20 | 12.80 | 2.20 | 33.30 |
| Pseudo (source geminates) | 0.00 | 0.90 | 0.00 | 0.40 | 0.00 | 0.90 | 0.00 | 1.40 |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |

**Table 7.15 : Minimum & Maximum percentages of Model D after lesioning.**

One method of achieving this is to make the non-geminate threshold a function of the number of geminates in the word as described in Equation 7.1.

$$T_{Ni} = T_N + G_i * \rho(\delta) \qquad \textbf{Equation 7.1}$$

where $G_i$ is the number of geminates in the word being presented, $T_{Ni}$ is the resultant non-geminate threshold for that word, $T_N$ is the standard lower threshold below which all non-geminate letters would normally be pushed, and $\rho()$ is a random function returning a value between $\delta/2$ and an upper value $\delta$. One benefit of the random function is that words containing the same number of geminated letters should have different propensities for generating pseudo geminate statuses.

Table 7.14 shows that selectively increasing the non-geminate threshold for words with source geminates, promotes non-doubled letters for these words to drift closer to the geminate threshold encouraging additions. This is not as prevalent in words without existing doubles and other than the counter example showed by AS, this behaviour has a qualitative match with LB and FM. Note also, that pseudo-additions for words without source geminates are higher than in words with source geminates. This behaviour is similar to the geminate addition behaviour shown by AS. This matches the behaviour shown by LB qualitatively. If we consider just ablation and noise, the general relative percentages of lost statuses and additions are comparable to patient LB. The constrain behaviour shows a good match with respect to additions to FM yet the

performance of the model is still not encouraging, especially related to geminate movement. Additions alone cannot be said to influence the movement of geminates to move. Typically, the addition would need to be accompanied by a corresponding delete, and is a key objective of Model E.

### 7.4.5 Model E

Influencing corresponding geminate deletes in the presence of an early geminate addition is achieved by slightly inhibiting, or *damping* the value of a letter's geminate activation following presentation of a double. This model prevents excessive *serial geminates* (more than one double in a row) that were seen to occur in models B, C and D, and implicitly provides a mechanism for geminate movement (or transposition). For this model, we use the same principle applied to letter transpositions discussed in Chapter 5. In other words, an incorrect production of a letter followed by its inhibition should tend to promote transpositions. We therefore assume that geminate movements may occur where a geminate is added too early in the word, followed by a subsequent damping of the valid geminate status, effectively bringing a geminate status forward by one position. Although geminate movement can occur via any geminate deletion and addition, this mechanism specifically caters for converting unexpected serial geminates into a geminate movement.

Unlike the approach by Glasspool & Houghton (2005), we do not prohibit serial geminates. Rather, we ensure that geminate activation following a double is very difficult to achieve unless it is very highly activated prior to damping. This allows the system to learn words in the English language such as TOFFEE, and BASSOON. During training, if a letter with a desired geminate has a preceding active geminate status, then we exaggerate the extent of reinforcement to be applied to the geminate by artificially increasing the target geminate activation. In Figure 7.4 for example the

original (or pre-damped) activation for the letter 'O' is trained to achieve a value such that damping does not affect its correct geminate status. A geminate is deemed to be active if according to Equation 7.2, its geminate status exceeds the geminate threshold. In other words, if

$$F(G)d > T_G \qquad \textbf{Equation 7.2}$$

where $F(G)$ is the logistic sigmoid function $F(x) = 1/(1+e^{-x})$ and $T_G$ represents the activation value of the geminate node. If a double was presented prior to the letter in question, then $d$ is equal to the damping value. Otherwise, $d$ is equal to 1.0. Figure 7.4 shows two activation series for the word 'BASSOONS'. The original shows the actual geminate activation values for each recalled letter. The second line shows the activation after applying damping of 0.80. Note than the 'N', is damped due to the production of the previous double 'O' even though it is still below the geminate presentation threshold. Unless a letter follows a double, geminate activation is unaffected.

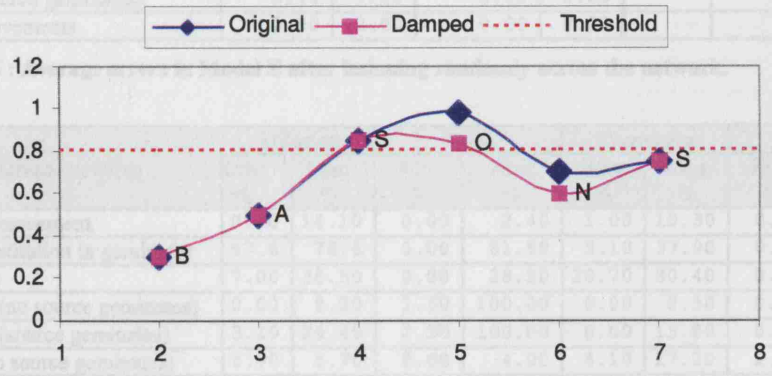



**Figure 7.4: Some potential natural and *damped* geminate activation values for the word BASSOONS**

Note that the damping value must always be greater than the geminate threshold. This can be seen by moving the damping value to the right hand side of Equation 7.2 arriving at Equation 7.3. This is logical since our activation function only has a range of 0.0 to

1.0. Any value of $d$ less than our geminate activation threshold would therefore require an activation function that could return a value greater than 1.0 to ensure the corpus could be trained.

$$F(G) > \frac{T_G}{d} \qquad \textbf{Equation 7.3}$$

One possible critique of artificially increasing the target activation for geminated letters following another geminate (e.g. the O in BASSOON) is that is seems somewhat contrived. We believe that our approach is defensible in the sense that it improves training times. Without this facility, the training approach would reach the same learned state anyway, albeit at a slower rate.

| Geminate activity | Ablation % | Noise % | Constrain % | Scale % | AS % | LB % | FM % |
|---|---|---|---|---|---|---|---|
| Geminate movement | 4.20 | 0.80 | 4.40 | 8.10 | 19.1 | 52.1 | 36.2 |
| Letter Substitution in geminates | 55.84 | 66.60 | 21.40 | 17.10 | 0.0 | 8.7 | 38.3 |
| Lost status | 22.88 | 12.50 | 62.50 | 54.10 | 28.6 | 20.2 | 21.3 |
| Additions (no source geminates) | 1.90 | 0.20 | 0.10 | 0.10 | 46.7 | 0.2 | 0.0 |
| Additions (source geminates) | 10.99 | 18.60 | 2.30 | 5.00 | 5.7 | 18.8 | 4.26 |
| Pseudo (no source geminates) | 3.80 | 1.20 | 9.20 | 15.60 | - | - | - |
| Pseudo (source geminates) | 0.40 | 0.10 | 0.10 | 0.00 | - | - | - |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | - | - | - |

**Table 7.16 : Average errors in Model E after lesioning randomly across the network.**

| Geminate activity | Ablation | | Noise | | Constrain | | Scale | |
|---|---|---|---|---|---|---|---|---|
| | Min % | Max % | Min % | Max % | Min % | Max % | Min % | Max % |
| Geminate movement | 0.00 | 14.10 | 0.00 | 2.40 | 1.00 | 10.30 | 0.00 | 33.30 |
| Letter Substitution in geminates | 47.6 | 76.8 | 0.00 | 81.90 | 5.10 | 37.90 | 0.00 | 37.50 |
| Lost status | 7.00 | 38.50 | 0.00 | 28.20 | 20.70 | 80.40 | 0.00 | 86.10 |
| Additions (no source geminates) | 0.00 | 8.20 | 1.50 | 100.00 | 0.00 | 0.50 | 0.00 | 1.40 |
| Additions (source geminates) | 3.10 | 24.40 | 2.50 | 100.00 | 0.00 | 13.80 | 0.00 | 50.00 |
| Pseudo (no source geminates) | 0.00 | 5.70 | 0.00 | 4.00 | 4.10 | 17.20 | 2.80 | 50.00 |
| Pseudo (source geminates) | 0.00 | 0.90 | 0.00 | 0.70 | 0.00 | 0.10 | 0.00 | 0.00 |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |

**Table 7.17 : Minimum & Maximum percentages of Model E after lesioning.**

The first observation regarding the performance model E is that it there is more geminate movement than with previous models. There are however, still too few to claim results comparable to patient data. Although geminate addition behaviour still seems to be a

good match to patients LB and FM, all three patients have show variability in the types of errors associated with geminate movement, which our model does not even approach. In addition, this model tends to accentuate the number of substitutions, in a way not consistent across all lesion types. Substitutions are very high with ablation and noise, yet low with scaling and constraint lesion types showing an overall range of 17.10% to 66.60%. Patient data shows a much narrower range (0.0% to 38.3%).

### 7.4.6 A Common Simulation Framework

Rather than use different source code for each model, we used the same program for each model, customising the learning and recall behaviour by modifying the program parameters listed in Table 7.18. Other than random seeds used to imitate quasi-patients, these are the only free parameters used in our simulations and we believe that this demonstrates a single framework addressing multiple behavioural models.

| Model | $T_{G(Learn)}$ | $T_{G(Recall)}$ | Margin | $T_N$ | $\delta$ | Damper |
|---|---|---|---|---|---|---|
| A | 0.95 | 0.80 | 0.025 | 0.05 | 0.00 | 1.00 |
| B | 0.80 | 0.80 | 0.025 | 0.77 | 0.00 | 1.00 |
| C | 0.80 | 0.80 | 0.025 | 0.60 | 0.00 | 1.00 |
| D | 0.80 | 0.80 | 0.025 | 0.60 | 0.15 | 1.00 |
| E | 0.80 | 0.80 | 0.025 | 0.60 | 0.15 | 0.85 |

**Table 7.18: Parameters used in each model**

## *7.5 A focal Lesioning approach*

Our approach so far in this chapter has been to explain geminate error behaviour using a diffuse lesioning strategy. In chapter 4, we described diffuse lesioning as damage to a large number of neural elements over a widespread area of the model. In the light of our results so far, it is worth evaluating the effects of focal lesioning on just geminate node connections, and whether this improves performance related to geminate movement. In these tests we took an intact Model E network, and applied various severities of ablation, and noise to connections leading to the geminate node (Table 7.19). Since lesions were

applied solely to geminate connections, there were no letter identity errors, and thus no possibility of letter substitutions, transpositions, inserts, or deletions. For example, without interspersed deletes between identical characters, there can be no pseudo geminates, and without letter identity errors, there can be no geminated substitutions. Therefore, of the original eight geminate error classifications, only four of these are listed.

| Ablation | | | | | Noise | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Amt. % | Posn Move % | Lost Status % | Adds (No Src) % | Adds (Src) % | Range | Posn Move % | Lost Status % | Adds (No Src) % | Adds (Src) % |
| 5% | **15.62** | 61.86 | 8.81 | 13.71 | -0.1→0.1 | **0.00** | 0.00 | 100.00 | 0.00 |
| 10% | **9.50** | 45.80 | 36.50 | 8.20 | -0.2→0.2 | **2.30** | 90.80 | 0.00 | 6.90 |
| 15% | **15.40** | 40.50 | 33.60 | 10.50 | -0.3→0.3 | **6.70** | 87.60 | 0.00 | 5.70 |
| 20% | **7.71** | 34.84 | 39.64 | 17.81 | -0.4→0.4 | **37.26** | **30.87** | **6.39** | **25.48** |
| 25% | **10.90** | 40.30 | 37.00 | 11.80 | -0.5→0.5 | **6.31** | 77.68 | 8.51 | 7.51 |
| 30% | **9.39** | 41.26 | 40.66 | 8.69 | -0.6→0.6 | **9.10** | 18.20 | 48.50 | 24.20 |
| 35% | **9.61** | 11.71 | 65.07 | 13.61 | -0.7→0.7 | **7.30** | 73.90 | 10.70 | 8.10 |
| 40% | **5.59** | 26.47 | 54.05 | 13.89 | -0.8→0.8 | **6.69** | 41.26 | 35.86 | 16.18 |
| 45% | **7.80** | 13.50 | 65.00 | 13.70 | -0.9→0.9 | **7.69** | 63.34 | 18.18 | 10.79 |
| 50% | **11.69** | 25.77 | 50.05 | 12.49 | -1.0→1.0 | **8.49** | 61.64 | 17.68 | 12.19 |

**Table 7.19 : Geminate Behaviour by ablating and adding noise to geminate related connections. Amt describes the percentage of connections to delete, and Range describes the range of noise to add to each affected connection.**

What seems apparent from Table 7.19 is that except for one lesion severity (random noise in the range –0.4 to 0.4), most other lesion types and severities had minimal influence on geminate movement. What should also be noted is that focal lesioning also had a direct influence on the types of geminate error. For example, words without doubles produced more geminates than words with doubles and this may be a consequence of the damping facility. In summary, it seems that a focal lesion strategy does not generally satisfy our desired behaviour for a working model.

## *7.6 A hybrid lesioning approach*

One final approach to establishing whether an augmented GSC model can satisfactorily explain geminate production is to combine focal and diffuse lesioning strategies. We

approached this by randomly adding noise in the range –0.4→0.4 to geminate node connections, and then applying ablation, noise, constraint and scaling lesions of various severities (see Appendix D) across and entire model-E network. Our motivation in attempting this is to ascertain whether the GSC model can viably explain patient behaviour albeit in a manner requiring more than a trivial lesioning method. The results in Table 7.20 are averaged over all lesion severities for fifteen quasi-subjects.

| Geminate activity | Ablation % | Noise % | Constrain % | Scale % | AS % | LB % | FM % |
|---|---|---|---|---|---|---|---|
| Geminate movement | 25.10 | 26.05 | 28.57 | 33.30 | 19.1 | 52.1 | 36.2 |
| Letter Substitution in geminates | 24.10 | 27.75 | 16.38 | 10.20 | 0.0 | 8.7 | 38.3 |
| Lost status | 10.30 | 4.29 | 25.08 | 22.30 | 28.6 | 20.2 | 21.3 |
| Additions (no source geminates) | 12.30 | 11.58 | 0.20 | 0.20 | 46.7 | 0.2 | 0.0 |
| Additions (source geminates) | 25.40 | 29.24 | 20.08 | 25.90 | 5.7 | 18.8 | 4.26 |
| Pseudo (no source geminates) | 2.40 | 0.70 | 8.79 | 7.70 | - | - | - |
| Pseudo (source geminates) | 0.40 | 0.40 | 0.90 | 0.40 | - | - | - |
| Pseudo movements | 0.00 | 0.00 | 0.00 | 0.00 | - | - | - |

**Table 7.20: Performance of Model E after applying focal and diffuse lesioning. Values are the average of all lesion severities.**

It is apparent that the combination of diffuse and focal lesion types shows better results. We have a general match of geminate movement results with FM and a good match of geminate additions to LB and FM with the constraint and scale lesion types. Substitutions show a more consistent range across all lesion severities than with diffuse lesioning of Model E, and scale and constraint show a reasonable match to LB with respect to most geminate error types. One exception is geminate movement as this is particularly high with LB.

## 7.7 An Alternative Architecture

Our initial approach to producing geminates involved extending the GSC model by adding a geminate status as an output value to be learned simultaneously with letter identity. Glasspool & Houghton (2005) also include a geminate node in their model (Figure 7.5), and their apparent success at producing plausible doubling errors highlights

that such a mechanism may not need to be as complex as ours has ended up. Their model however, differs in a number of respects. Firstly, although their geminate node is located in a similar position to its equivalent in our model, there is no word identity as each corpus member is given its own model. Secondly, the item layer is effectively a proxy representation for the values produced by the IE nodes. Their description (p311), states that "*Each item node acts as a place-holder for a different response. During recall, an item node achieves its maximum activation when the Initiate and End node activations are in the state they were in at the corresponding sequential position during learning*". In our model, the context (such as IE or moving window) is the only mechanism providing a temporal order to constituent letters. Our second layer for example is a hidden layer. Thirdly, the model is localist insofar as every corpus member is given its own model thus avoiding issues of crosstalk associated with conflicting serial sequences associated with multiple words.

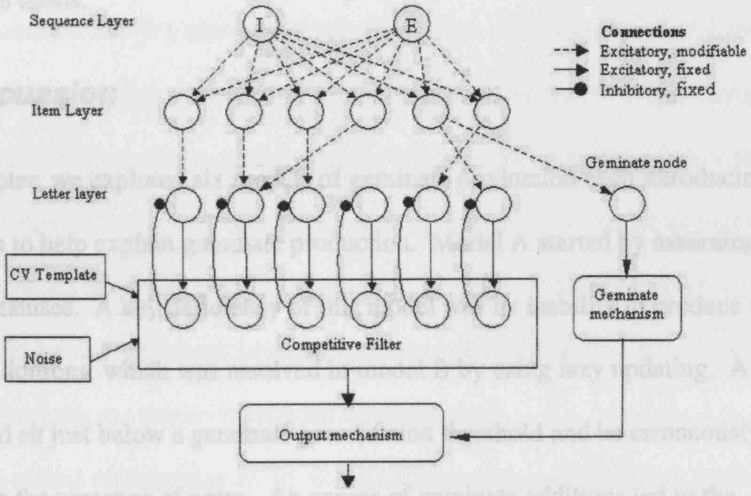



**Figure 7.5: Glasspool & Houghton's (2005) model simulating geminate and CV behaviour.**

One possible extension to our model is to provide a completely separate hidden layer used solely by the geminate node (see Figure 7.6). Geminate output is a single value so

a hidden layer of only two nodes may be sufficient. Separating the hidden layer to be independent of any interaction with graphemic identity also accords with an independent geminate production mechanism.

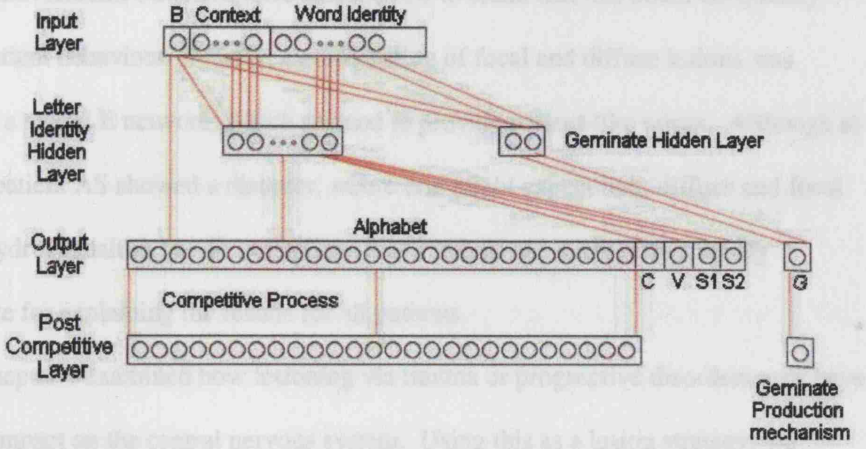



**Figure 7.6: Alternative Graphemic Buffer architecture with an independent geminate hidden layer.**

We believe that the proposed architecture has merit, but we consider it to be beyond the scope of the thesis.

## 7.8 Discussion

In this chapter, we explored six models of geminate production each introducing a new mechanism to help explain geminate production. Model A started by assuming binary geminate statuses. A key deficiency of this model was its inability to produce sufficient geminate additions, which was resolved in model B by using lazy updating. A Geminate status could sit just below a geminate presentation threshold and be erroneously activated in the presence of noise. An excess of geminate additions led to the introduction of the non-geminate threshold in model C. Unfortunately, there was no selectivity, in whether words with existing geminates showed more doubling errors than words without. A word specific non-geminate threshold in model D catered for this and

provided a slightly better fit to the patient data. All models seem to produce too few geminate movements and one objective of Model E was to introducing a damping mechanism to promote geminate transpositions. This did increase the overall number of geminate movements, but there were still too few to claim that this could adequately explain patient behaviour. Finally, a combination of focal and diffuse lesions was applied to a model E network, which seemed to provide patient-like errors. Although at least one patient AS showed a disorder, where one might expect both diffuse and focal lesions (hydrocephalus), we do not believe that a hybrid approach is necessarily appropriate for explaining the results for all patients.

Chapter 4 examined how lesioning via trauma or progressive disorders may have a diffuse impact on the central nervous system. Using this as a lesion strategy, we attempted to show that diffuse lesioning could explain common symptoms of geminate disorder. Unfortunately, to mimic patient results, it was also necessary to focally lesion connections leading to the geminate node as well as applying specific lesion ranges on geminate connections. We also investigated the effect of a broad range of lesions, which in many cases proved too wide or too narrow. It is possible that too little damage simply had insufficient impact on the geminate node, and too much damage led to a general breakdown of the network such that plausible geminate behaviour was unsupported. Our model, or our lesioning approach is therefore open to criticism, but we would however claim that we have not been prescriptive in our lesion strategies or severities and that our results are based on multiple quasi-patients.

The results presented so far in the thesis suggest that the model is more prone to substitutions than transpositions, so we might therefore expect geminates to behave in a similar fashion. In other words, we should not expect many geminate transpositions. Further, our distributed approach did not connect the context mechanism directly to the

geminate node in the same way as Glasspool & Houghton's localist network. A hidden layer separates our context and letter production systems, whereas the context via the item layer seemed to promote geminate movements more than our model.

Clearly, the deficiencies in our model may imply that geminate production needs to be rethought, and in section 7.7 we suggested a possible alternative architecture. Whether this alternative approach produces more plausible behaviour or not, we would argue that the process of progressing from model A to model E has provided a number of mechanisms that may be of use in other models. For example, Glasspool & Houghton prohibit serial doubles by strongly inhibiting the geminate feature node. This may be appropriate for many words, but it is an unrealistic constraint to apply to the English language, as words such as BASSOON and TOFFEE could never be learned. Sequential geminates do not seem to be an English specific requirement, so for the model to provide a plausible cross-language mechanism, strict geminate inhibition seems somewhat restrictive. The use of a damping mechanism provides a way of achieving this in our model, and may also be appropriate in their model resolving one of their own critiques

One possible reason for fewer geminate movements may be attributed to a more conservative scoring mechanism. For example, PRINCESS → PINTESS could be classified as a geminate movement as the geminate is clearly in a different position. Our automatic analysis however, classifies this as a consonant deletion ('R') in position two, followed by a consonant substitution ('C'→'T') in position five, concluding no geminate related error behaviour. Although our results produce many similar examples with geminate movement being avoided through interpreted deletes or additions, automatic analysis of patient FM's raw error data clearly shows many geminate movements. The

lower geminate movement count therefore seems to be due to an inherent weakness in the model, and not the scoring mechanism.

In evaluating the overall approach, we considered that one can always *tune* parameters and lesion a model to produce results that will match patient data, exposing the modeller to a critique of "curve-fitting". However, we have tried to mitigate the risk of such a critique by exploring a combination of diffuse and focal lesion types. Although the most plausible geminate behaviour is limited to a narrow range of lesioning, it does seem to have been done in a defensible manner. Pure models B, C and D produce a number of symptoms shown by patients, and the hybrid approach (Section 7.6) with intact models B, C, and D seemed to extend the range of suitability to patient data. This chapter does not describe in detail, the various combinations of geminate and non-geminate thresholds, damping factors, and values for $\delta$ that were investigated prior to arriving at our final values. However, alternative values did not seem to produce a marked qualitative improvement in the performance of our models, and locating the perfect combination of parameters would only have shown that one can indeed fit the data to the model. It may be worth considering a more extensive study to establish ranges and parameter combinations that do indeed provide better results, under which conditions these exist, and whether these are plausible foundations for a model, but we consider this beyond the scope of the thesis.

This chapter is the last detailing the results of the simulations. In the final chapter, we will summarise the thesis highlighting positive and negative aspects of the model, and where we believe future research may be of benefit.

# 8 Discussion and Future Work

## *8.1 Introduction*

In this thesis we followed an approach of taking an existing model of spelling (Glasspool et al, 2006) that used connectionist networks to represent a semantic system and graphemic output buffer (GOB) and developing it further. Symptoms of graphemic buffer disorder (GBD) and deep dysgraphia (DD) have been attributed to damage in the GOB and semantic systems respectively, and the model tested the hypothesis that selectively damaging each sub-network would produce different attributes associated with each symptom. The error distributions or the shape of the serial position curve for example, were predicted to change as a function of lesion location, thus reflecting the hypothesised locus of impairment in the spelling model. In augmenting the model, we examined the original architecture making a number of changes which we believed could be justified on theoretical or practical grounds. We also provided a workbench allowing us to systematically analyse test results after applying lesions to specific network locations. One conclusion we arrived at, is that the original model produced the behaviour claimed by Glasspool et al, but was not always robust in the presence of damage when using a more systematic testing procedure.

This chapter summarises a number of topics where our investigations lead to surprising results. Either, the model did not behave as expected in the sense that testing exposed some weakness, or explorations using the new model highlighted areas of behaviour that have not been modelled before.

## 8.2 Related Putative Functional Syndromes

In chapter 3, we examined in some detail, the symptoms of Graphemic Buffer Disorder (GBD), and Deep Dysgraphia (DD), and discussed Cipolotti et al's (2004) claims, firstly that these patterns of behaviour can be viewed as putative functional syndromes, and secondly, that there is a relationship between them. We provided a more extensive literature review broadly grouping patients into two categories; those presenting with symptoms associated with damage presumed to exist solely in the graphemic buffer (GOB), and those with damage presumed to exist earlier in the lexical-semantic pathway (NON-GOB). We concluded that the Cipolotti et al claims were robust in the sense there does indeed appear to be a relationship between both syndromes. This observation was however qualified by the fact that some type A and B characteristics, as Cipolotti et al termed them, cannot be measured in the context of single errors alone. Their conclusions are thus difficult to corroborate using many of the published results. With respect to the simulation however, the model seems to provide a plausible explanation for the behaviour of single errors.

There also seems to be a consistent relationship between locus of impairment and the nature of the serial position curve. The presence of monotonically increasing serial position curves with concomitant deletes can be explained by suboptimal GOB performance since the GOB is responsible for both the selection and serial ordering of constituent letters. With no clear evidence of damage to the GOB, the possibility that serial position symptoms typically assumed to relate to damage to the graphemic buffer may indeed be related to damage presumed to exist earlier on. The simulations described in Chapter 6 clearly showed that damage applied prior to network A (i.e. the Graphemic Output Buffer) did result in monotonically increasing serial position curves, and a majority of deletes (i.e. type B). For patients who show symptoms of both DD and

GBD, this suggests multiple loci of impairment, so one cannot conclude that errors necessarily arise as a result of a functional relationship between syndromes. With this in mind, we categorised our patients according to their predominant symptoms. As far as we could ascertain however, GOB and NON-GOB patients seemed to fit type A and B behaviours respectively.

We also identified one possible extension to their type A and B characterisations, namely the steepness of the word-length effect. Patients with damage presumed to have occurred within the GOB typically produced word-length curves with a significantly steeper gradient than patients where the GOB was apparently intact, and damage was presumed to exist earlier in the spelling mode. The reason for this behaviour may relate to the ability of the GOB to *cope* with degraded input far more efficiently than had it been damaged. A working buffer when presented with degraded input is conceivably going to perform better than a damaged buffer. Graceful degradation is a key attribute of computational and biological connectionist systems, and such systems are expected to behave as normally as possible. Clearly, a damaged GOB would be expected to show a word-length effect, but in the case where the GOB is exposed to degraded input, one might argue that excluding *damaged* input, the serial production mechanism is generally intact. The model allows such a theory to be easily tested.

In order to validate whether a similar behaviour was produced by the model, we examined the relative resultant word lengths gradients after lesioning network A, and network B in various locations. Table 8.1 shows an indicative comparison between one network A (A-MW) and one network B (B-DGSB); a complete comparison is provided in Appendix I for all networks. As with previous tests, each result is based on the average of our 15 quasi-patients. A Mann-Whitney test comparing gradients in Table 8.1 shows a significant difference between the two network types ($p < 0.001$). Using all

available data for all A and B networks also shows a significant difference ($p < 0.001$). This seems to behave similar to results we found in patient data (see Chapter 3).

| Lesion Location | Lesion Type | Network A – A-MW | | | | | Network B – B-DGSB | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Word Length | | | | m | Word Length | | | | M |
| | | 4 | 5 | 6 | 7 | | 4 | 5 | 6 | 7 | |
| Random | Ablate | 39% | 51% | 59% | 65% | **0.09** | 58% | 63% | 66% | 67% | **0.03** |
| | Noise | 35% | 47% | 54% | 59% | **0.08** | 35% | 40% | 43% | 45% | **0.03** |
| | Constrain | 32% | 40% | 48% | 51% | **0.07** | 43% | 49% | 53% | 53% | **0.03** |
| | Scale | 27% | 32% | 38% | 40% | **0.05** | 19% | 31% | 38% | 40% | **0.07** |
| Input to Hidden | Ablate | 42% | 56% | 66% | 70% | **0.09** | 49% | 53% | 57% | 57% | **0.03** |
| | Noise | 27% | 40% | 48% | 52% | **0.08** | 38% | 42% | 46% | 46% | **0.03** |
| | Constrain | 27% | 39% | 48% | 51% | **0.08** | 58% | 61% | 65% | 64% | **0.02** |
| | Scale | 31% | 43% | 51% | 54% | **0.08** | 26% | 33% | 37% | 35% | **0.03** |
| Context to Hidden | Ablate | 46% | 60% | 69% | 74% | **0.09** | **N/A** | | | | |
| | Noise | 25% | 36% | 44% | 51% | **0.09** | | | | | |
| | Constrain | 29% | 41% | 49% | 53% | **0.08** | | | | | |
| | Scale | 36% | 46% | 55% | 58% | **0.08** | | | | | |
| Word Identity to Hidden | Ablate | 47% | 62% | 73% | 78% | **0.10** | | | | | |
| | Noise | 33% | 45% | 53% | 57% | **0.08** | | | | | |
| | Constrain | 17% | 35% | 49% | 60% | **0.14** | | | | | |
| | Scale | 15% | 33% | 48% | 59% | **0.15** | | | | | |
| Hidden to Output | Ablate | 38% | 51% | 62% | 67% | **0.10** | 41% | 45% | 46% | 48% | **0.02** |
| | Noise | 33% | 44% | 51% | 56% | **0.08** | 31% | 35% | 39% | 42% | **0.04** |
| | Constrain | 26% | 29% | 32% | 34% | **0.03** | 28% | 33% | 37% | 40% | **0.04** |
| | Scale | 47% | 50% | 50% | 47% | **0.00** | 21% | 27% | 33% | 39% | **0.06** |
| Output | Noise | 29% | 42% | 50% | 55% | **0.09** | **N/A** | | | | |
| | | **N=21** | | **Average** | | **0.08** | **N=12** | | **Average** | | **0.04** |

**Table 8.1: A comparison of network A and network B word length effects and their associated gradients. The percentages relate to total errors produced by each respective word length, and the *m* value describes the associated gradient calculated using least-squares regression.**

Despite the statistical significance of our analysis of patient data, we expressed some reservations about the strength of our conclusions as they were based on the results of only seventeen patients. Clearly, further analysis using data from a larger number of pure GBD and DD patients may provide more conclusive evidence of a difference in word-length effect. The fact that our model also produces a similar effect however gives us some confidence that further investigations should confirm our initial findings.

## 8.3 A New Lesion Technique

In Chapter 4, we summarised a number of effects seen in the presence of damage to the nervous system, making connectionist analogies where possible. One aim of building models of nervous system activity is to determine whether the model behaves plausibly,

not just in terms of its outward behaviour, but also with respect to the design principles underlying the model. This is often difficult, firstly because of the complex interactions between underlying processes in the nervous system, and secondly that interpreting data from simplified models may be difficult to substantiate. We summarised a number of commonly used lesion types (such as ablation and noise), but also introduced what we believe is a novel lesion type.

This *constrain* lesion mimics the effect of progressive demyelinisation, remyelinisation and a concomitant deterioration in connectivity often seen in disorders such as Multiple Sclerosis. Our tests seemed to show that the constrain type behaved similarly to the scale lesion type in the sense that both lesion types effectively reduced overall activation function values, whereas ablation and noise lesions result in fluctuations of the overall contribution to an activation function. We would conclude that ablation and noise allow the networks to degrade more gracefully than the scaling and constraint lesion types. This could be seen by the actual versus expected behaviour of both networks. For example, in tests of frequency for network A (see Chapter 6), the network had a tendency to produce anomalous results where the lesion type was scale and constraint; high frequency words were more erroneous. Similarly, in tests of simple versus complex CV error rates, scale and constrain types tended to produce fewer errors for complex than simple CV words for both network A and B. Indeed, constraint and scale lesions were also more likely to produce *no* significant effect of CV complexity. This behaviour must also be considered anomalous if error rates for simple CV words are expected to be lower than for complex CV words.

In summary, the effects seen with scale and constrain lesion types may suggest that they are more appropriate when modelling *substantial* damage to a network, over

and above that where graceful degradation may be expected to provide feasible, if erroneous behaviour.

## 8.4 Semantic Network Design

In Chapter 6 our tests on concreteness showed that network B (the semantic system) was susceptible to the design of the semantic feature vectors to the extent that behaviour contrary to that produced by patients could be consistently produced. An investigation of the underlying hidden unit activity (Appendix H) suggested that the Glasspool semantic representations allowed both abstract and concrete words to be less prone to error under the right conditions. Clearly, in order to behave consistently with general patient behaviour, concrete words must consistently be more robust in the presence of damage.

Using a variation of Plaut & Shallice's (1993) design produced a consistent concreteness effect across a wide range of lesion severities, locations, and types. Glasspool (personal communication) placed no theoretical weight on the design of network B in the sense that damage to network B was not necessarily expected to behave completely *plausibly*. Indeed, the method used to investigate the relationship of the model to putative functional syndromes as far as the original paper by Glasspool et al (2006) centred around "*explaining the most salient differences between the two syndromes, which are seen as consequences of the difference between an intact sequence generation system operating on degraded input versus a damaged sequencing system operating on intact input*". The *type* of degraded input however, seemed to be less important than the fact that it was *degraded*. This approach seemed to perform adequately, and indeed, had we not applied a more rigorous methodology to testing the effect of semantic representations on error rates, the deficiency in the approach may not have been as evident. Clearly, more thought may be required for a semantic vector

design if further investigations involving both network A and B are to be considered, and we discuss this in more detail toward the end of the chapter when describing a potential future architecture.

## 8.5 Testing Approach

In presenting our results in Chapter 6 and Chapter 7, we attempted to mitigate the risk of these being biased by the network's starting state as a result of a specific random seed. We trained three networks with random seeds, and then tested each trained network by randomly lesioning using five random seeds. Each random seed sets a different starting state for the random number generation, thus providing different behaviour for each network. Thus, where possible our results were based on an average of fifteen *quasi-subjects.* A model may also be subject to critique if the lesion ranges applied to produce patient-like symptoms are too prescriptive. For example, if ablating 1.5% of the network provides *good* results, and ablating less than 1.4% or more than 1.6% of the network provides *bad* results, then it is difficult to claim that the model fits the patient behaviour under more general conditions. In our tests, we applied ten progressively increasing lesion severities to our fifteen quasi-patients thus providing us with 150 instances of disordered behaviour. These were then averaged to give us a general indication of whether the model was able to produce behaviour in line with patient data. Our analysis of the concreteness effect using this approach highlighted that by applying systematic damage, the Glasspool semantic design did not consistently produce results reflect those of patients.

## 8.6 Comparing Serial Position Mechanisms

There are at least two approaches to controlling and influencing serial position such that in the presence of damage, medial letters tend to be more erroneous than at either end.

The first by Houghton (1990) used Initiator/End (IE) values and the second by Burgess & Hitch (1992) used a moving window (MW) of nodes to direct the serial positions associated with a word's constituent letters, both of which were designed to introduce a level of positional ambiguity in medial letters. These serial mechanisms or contexts are potentially an intrinsic part of the model so we examined how the choice of each may have influenced error distributions, and the ability to conform to the effects of concreteness, frequency, and CV complexity.

We also compared the performance of each of these contexts to a third *control* context, which was similar in design to the MW context, albeit with a window size of one. With no overlapping serial component, we believed that the control context would show no serial position affect resulting in a flat error curve. This control context was termed the *positional* context due to the fact that each constituent letter had a unique position within the context. Our tests did indeed show that both standard contexts produced the expected bow-shaped serial position curves, and roughly similar error distributions.

The IE context seemed more robust to damage than the MW context in the sense that the IE context showed no anomalous effects of concreteness, frequency, and CV complexity. In each of these tests, the MW context produced instances of substantial significant differences between abstract and concrete words, and low and high frequency words. This effect was never seen with the IE context, suggesting that the IE context produces the effect required of a network simulating the GOB more consistently. Contrary to expectations however, the positional context did not produce a flat serial error curve, but one showing fewer errors as a function of serial position. This was surprising and may be due to the inhibition algorithm used by network A. As discussed in Chapter 5, the role of inhibition is to remove a letter from the competitive process in

the serial position immediately following its production. Inspiration for this mechanism is based on neurological evidence to suggest that when a synapse fires it cannot fire again for a brief period (e.g. Dayan & Abbot, 2001). There is evidence (e.g. Warrington & McCarthy, 1983, Warrington & Cipolotti, 1996, Gotts & Plaut, 2002) to suggest that access/refractory pattern exists across semantically related stimuli. In other words, when a stimulus is semantically related to a prior stimulus, then synaptic depression can affect many of the same neurons to the extent that patients make more errors than were the stimuli semantically distant.

How therefore does the inhibition algorithm produce a serial position curve which is not flat? Removing a single letter from the competitive process reduces the number of letters available to participate in errors. This seems obvious, but what may not be so obvious is that in say position five of a word, there are up to four letters that will be inhibited to varying degrees from participating in the competitive (and hence error) process, and may explain how fewer errors exist at late serial positions. With the IE and MW contexts, it is easy to see how this effect was obscured by a bow-shaped curve. Later in this chapter, we investigate possible improvements to the inhibition algorithm.

We believe that one artefact of the positional context is the presence of progressively fewer errors as a function of serial position, and a similar, albeit more pronounced effect is often seen in patients with unilateral neglect. In tests with network B however, progressively more errors as a function of serial position seemed to result from a lack of activation in word identity nodes passed from to network A. We do not claim that neglect and the serially increasing error curve have similar causes, but it was of interest to investigate an approach to lesioning the IE context so that one could systematically produce serial position curves similar to those found in left and right side neglect. This involved modifying the standard I and E functions in such a way that

damage could be selectively added to either term. Deep dysgraphia requires damage to both the lexical and non-lexical paths, and one could argue that the serial effect seen in DD patients need not be an indirect effect, but could be caused by a specific type of damage to the GOB in conjunction with multiple loci of impairment across the spelling system. Cubelli (1991) for example, argued that the GOB itself may be selectively damaged referring to an inability to produce vowels. It is also interesting to note that selectively lesioning either the I or E components also produced a majority of deletes in most cases.

## *8.7 Orthographic Complexity*

It has been suggested that graphemic representations are organised in dimensions or *tiers* of information (e.g. Caramazza & Miceli, 1990, McCloskey et al, 1994, Buchwald & Rapp, 2003). In a multi-tier model, each tier theoretically identifies different aspects of a word's constituent elements, such as consonant/vowel status, geminate status, letter identity, and possibly even syllable membership. In Chapter 6, we examined the effect of the CV nodes in network A. It was clear that without these nodes, error behaviour was similar to chance in that consonants and vowels were likely to interchange (substitute or transpose) as a function of their relative frequencies in the corpus. By adding CV nodes however, more within-class (e.g. vowel for vowel) errors were produced.

The model also produced significantly different error rates for simple and complex CV words, and demonstrated that this can arise as a consequence of orthographic structure alone. Despite the critique made by Jonsdottir et al (1996), the position of Caramazza & Miceli (1990) is still considered viable, and there is an obvious argument for support for the graphosyllabic structure in that simple CV words were certainly less erroneous than complex CV words in their study. This was the first major

finding in Caramazza & Miceli (1990), and can be accounted for by their model of the organisation of the orthographic system. Caramazza & Miceli then go on to suggest that orthographic syllable representations provide the underlying effect. We believe this inference cannot be drawn from the data, since a similar explanation may be made through reference to a minimum complexity principle. In our model at least, this effect cannot be due to phonological or syllabic mechanisms since none exist, so the results can only be attributed to the inter-relationships between grapheme identities. Orthography alone could account for the fact that Italian patient LB produced far fewer errors on simple CV than complex CV words, which accords with the performance of the model. This effect was not however seen in English patients AS (Jonsdottir et al, 1996), and JH (Kay & Hanley, 1994) and it is therefore possible that language may be a factor in the different behaviours.

Analysing the raw data for patients AS and BA showed that while neither patient showed more errors on complex than simple CV words, each seemed to produce errors in line with a minimum complexity principle where the CV structure of an erroneous word was simpler than the target structure. This effect was seen in 86 of 105 tests (82%) using different lesion locations and types across word lengths from four to seven letters, with the effect stronger in shorter words. Defining a quotient allowed us to analyse how different these CV complexities were, in a less subjective fashion. We also noted that Caramazza & Miceli (1990) claimed evidence for a syllabic tier, based on tautosyllabic errors. They suggest that letters within the same syllable (tautosyllables) should be more inclined to transpose than across syllabic boundaries. By analysing their examples, it seems that a minimum complexity principle is just as likely to account for this, with transpositions more likely to occur at shorter than longer distances.

## 8.8 Error Distributions

The GSC model tends to produce many of the error distributions shown by patients, and for many there is a good qualitative match. Depending on lesion location and type, the majority of errors produced by the model were either substitutions or deletes with fewer transpositions and inserts. While this seems to reflect the behaviour of many patients, the model cannot explain the behaviour of those showing many more inserts. This deficiency has already been noted (e.g. Glasspool & Houghton, 2005, Glasspool et al, 2006), and the option of adding more than one letter in a single time slice has even been considered under the right circumstances (Glasspool & Houghton, 2005). In Chapter 6, we examined how modifying the letter threshold affected error distributions and assumed that by altering the threshold on network recall this may allow more inserts to occur. By Raising and lowering the threshold, many error distributions were produced, which were qualitatively similar to six patients (MRF, LB, DH, CW, ML, and SE).

Contrary to our earlier approach of producing different distributions via different lesion types in various lesion locations, this technique produced a wide variety of distributions using a single lesion type and location and solely modifying the letter threshold. As modellers, we are aware that altering free parameters for the purpose of fitting results to behaviour is risky. It is too easy to assume that the model's internal structure reflects those of patients, when in fact it is the apparent external behaviours that seem to match. If this experiment had produced more plausible quantities of inserts, it might, have been an indication that the existing architecture is capable of producing a broader range of errors, and that considering the letter threshold as *lesionable* may be a viable strategy. After all, the effects of neuromodulation serve to increase the sensitivity (or not) of neural circuits to input, and moving the threshold up or down would be comparable to damping or accentuating the activations produced by the output layer.

## 8.9 Geminates

The GOB is deemed to influence the behaviour of doubling errors, yet the GSC model did not investigate this at all. Further work by Glasspool & Houghton (2005) did use the principles of a competitive queuing model to produce geminate errors, but this was not a comparable test since each word was given its own network, and geminates were linked directly to a serial position mechanism. Using the extended GSC model described in Chapter 5, we added six different mechanisms, each explaining aspects of geminate behaviour. An evolution of increasingly complex approaches showed that the model could explain the majority of patient geminate errors. We also provided a means for resolving a deficiency described in Glasspool & Houghton's model of being unable to cater for serial geminates (e.g. BASSOON, TOFFEE), as well as a mechanism for producing more geminate errors on words with doubles than on words without doubles. One clear deficiency in the models seems to be a relative scarcity of geminate movements. Glasspool and Houghton's model showed a better fit to the patient data than our model, but as described earlier required the geminate node to connect directly to a serial position mechanism. Further modifications in our model may resolve this.

## 8.10 Future Work

Throughout the thesis, we identified a number of areas where further development of investigation was deemed to be beyond the scope of the thesis. We now expand on these:

### 8.10.1 The Graphosyllabic Tier

As described in 8.7, Caramazza & Miceli (1990) claimed evidence for a graphosyllabic tier, which they justified on the basis of tautosyllabic errors. Although their examples seemed to substantiate this claim, the same behaviour also seemed to conform to the minimum complexity principle. The GSC model uses distinct CV nodes to flag each

letter's CV status, and in our approach to implementing geminates, we similarly flagged each double letter with an active geminate status resulting in two instances of a letter being produced where required. Assuming that patients do indeed show consistent tautosyllabic errors, further status nodes could be added where each constituent letter is also associated with a syllable identifier. Using CV nodes assisted consonants and vowels to substitute and transpose with other consonants and vowels respectively, and we believe that syllable identity would similarly transfer to tautosyllable members.

### 8.10.2 The 'Unknown' Threshold

In Chapter 5, we described the stop threshold and letter thresholds. In cases where the winning activation is lower than the stop threshold, spelling ceased. Where the winning activation is higher than the letter threshold, that letter is produced, and if geminated, is produced twice in the same time slice. Patients have been reported (e.g. Katz 1991, Cubelli, 1991, Nolan & Caramazza, 1983, Miceli, Silveri & Caramazza, 1985) who leave blank spaces between correctly written letters. The patient may be aware of errors, but not necessarily know *which* letter belongs there. This suggests that a simple letter threshold may not truly reflect patient behaviour, and that catering for *unknowns* warrants further effort. Unknown letters have been classified as deletes (e.g. Nolan & Caramazza, 1983), but we believe there is merit in measuring unknowns distinctly from deletes thus distinguishing between patients unable to choose the target letter (e.g. TASTE → T_ST_) and those making incorrect choices leading to substitutions (e.g. TASTE → TOSTA). An unknown threshold $T_u$, situated between the presentation and stop thresholds, $T_r$ and $T_s$ would allow the system to be aware that a letter required presentation, but was not active enough to be identified, thus presenting a blank or a '?' character. In experiments not detailed in the thesis, we did indeed achieve the desired

effect, and altering the unknown threshold up or down produced varying distributions of unknown letters. Patient data is very sparse with respect to unknowns, so there was little opportunity to make a true comparison between the model and actual behaviour. The model does however seem to produce the required behaviour.

### 8.10.3 Letter specific thresholds

The ability to add an unknown threshold showed that the model is quite flexible in terms of the range of error behaviour resulting from a simple change. We also allow each individual letter to have separate thresholds. For example, the letter threshold for vowels may be different from consonants, and this may be justified when modelling the performance of patients who show clear differences in their error rates associated with consonants and vowels. By making vowels harder to learn, we may find behaviour similar to that described by Cubelli (1991) resulting in an inability to produce vowels. As we saw in Chapter 5, increasing the letter threshold results in more deletes, but doing so for specific letters makes them (e.g. vowels) more prone to omission. This facility already exists in the model but has not been tested extensively. Throughout our experiments, every letter's threshold is given the same value.

### 8.10.4 Production of Semantic Errors

As described in Chapters 5 and 6, a core consideration in designing semantic feature vectors is the effect such designs may have on the ability to produce a viable concreteness effect. Clearly the production of semantic errors such as TIME→CLOCK (Bub & Kertesz, 1982) are needed if other symptoms of deep dysgraphia are to be modelled. In the conclusion of Chapter 6, we described our investigations into using a semantic vector where attributes for words with a related meaning were shared. These vectors were used as input to network A, and showed that in principle, instances where

a word in error is semantically related to the target word is a viable output of the model. This has been seen in a number of patients such as BA (PUDDING → DESSET, SPAIN → MEX, CABINET → CUPBROAD), and patient TH, (ARTICHOKE → CAULFLOWER). In deep dysgraphia, the lexical-phonological route is damaged, so one can plausibly pass pure semantic input via a lexical output buffer into the GOB. In our tests, we passed semantic input directly to the GOB. This is theoretically implausible, but it did demonstrate that the model was able to produce errors similar to those produced by BA and TH.

In a revised model (see Figure 8.1 on page 255), we consider dividing network B into three sub-networks called B1, B2, and B3. Networks B1 and B2 would act as a rudimentary semantic system and graphemic output lexicon respectively, and network B3 connects each. For B1 and B2, we believe that self-organising networks (Kohonen, 1995) will allow semantically and orthographically similar items respectively to collocate within each network. Network B3 would be a simple back propagation network. The spatial proximity of semantically and orthographically similar words would be expected to produce semantically related errors (e.g. Bub & Kertesz, 1982), and orthographically related errors (e.g.McCloskey et al, 2006) respectively. Hinton & Shallice (1991) provided a model in which a critical property is *basins of attraction*, and we would expect that proximal items would have a higher chance of being interchanged in the presence of network damage.

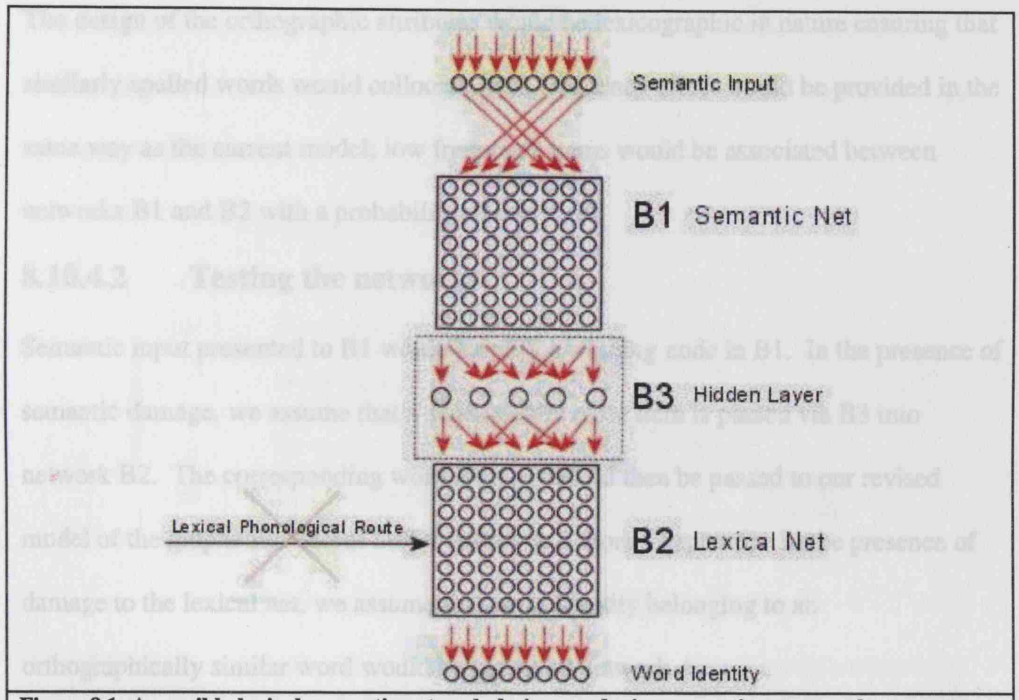



**Figure 8.1: A possible lexical-semantic network design producing semantic errors and concreteness and frequency effects. Network B1, and B2 are Kohonen self organising networks, and network B3 is a back propagation network.**

### 8.10.4.1 Training the networks

Each corpus member will have a semantic and an orthographic representation. To initially train the model, every corpus member will be presented to each network independently using its respective representation. For each network, this process is continued until there is a natural semantic or orthographic collocation of similar items, after which, network B3 would then relate each semantic vector to an orthographic equivalent. Note that in this model, there is no input from the lexical-phonological route, which is to be expected from deep dysgraphia. The design of the semantic vector could follow a similar approach to that used by Plaut & Shallice (1993), in which semantic attributes are given the values ON or OFF depending on whether that word represents something with those characteristics (e.g. *has-legs*, *is-alive*). We assume that concrete items will have substantially more ON attributes than abstract items, and that this should provide the concreteness effect expected from damage prior to the GOB.

The design of the orthographic attributes would be lexicographic in nature ensuring that similarly spelled words would collocate. The frequency effect would be provided in the same way as the current model; low frequency items would be associated between networks B1 and B2 with a probability of only 0.30.

#### 8.10.4.2 Testing the networks

Semantic input presented to B1 would identify a *winning* node in B1. In the presence of semantic damage, we assume that a semantically *close* item is passed via B3 into network B2. The corresponding word identity would then be passed to our revised model of the graphemic output buffer (network A from Chapter 5). In the presence of damage to the lexical net, we assume that word identity belonging to an orthographically similar word would be passed to network A.

#### 8.10.4.3 Revised Inhibition Algorithm

As described earlier, the inhibition mechanism prevents a letter from being available in the serial position immediately following its production, and is a requirement for a CQ approach. This is consistent with how the model produces geminates, since without a specific doubling mechanism it would be unable to produce them. Glasspool's original inhibition mechanism forced a winning letter's activation to be negative, which we believed went counter to the nature of the activation function having a value between 0.0 and 1.0 and we therefore modified this approach by dynamically scaling a letters activation value relative to how recently it was produced (see Gotts & Plaut, 2002 for a comparable approach). Such a function reflects the recovery pattern of a neuron, which has just fired becoming progressively more available over time. We believe that this gradual recovery mechanism may be indirectly responsible for an artefact seen in our analysis of serial position curves. Where the IE and MW contexts were expected to produced bow-shaped curves, the positional context was expected to produce a flat

curve with errors equally likely in any position. Figure 8.2 shows how error rates consistently decrease for all word lengths for later serial positions for a network trained with the control context.

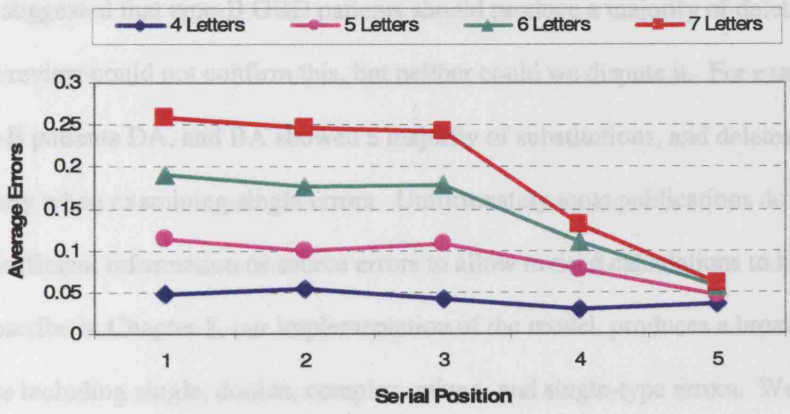



**Figure 8.2: Adding noise to the output layer of network A, trained using the positional context shows how error rates decrease as a function of serial position for words of lengths 4, 5, 6, and 7. Average errors are normalised across five serial positions using the modified Wing and Baddeley (1980) approach – See appendix B.**

It is open to question whether immediate (as opposed to gradual) recovery of an inhibited letter is biologically plausible, but this would seem to avoid the indirect effect of making fewer letters available to participate in erroneous behaviour as a function of word length.

## 8.11 Conclusion

This thesis took an existing model of the GOB augmenting it for the purposes of achieving four objectives. Firstly we would subject the augmented model to a more rigorous testing procedure than the original, highlighting areas of architectural instability. We believe this was achieved through the combined use of quasi-subjects, multiple lesion types, and a broad range of lesion severities.

Secondly, we investigated further Cipolotti et al's (2004) claim that that a putative functional relationship exists between GBD and DD. A broad literature review

did indeed suggest that both putative functional syndromes seemed to share common attributes. One of the difficulties faced with validating this further stems from the fact that the literature often publishes patient data in terms of single errors. Cipolotti et al for example suggested that type-B GBD patients should produce a majority of deletes. Our literature review could not confirm this, but neither could we dispute it. For example, two type-B patients DA, and BA showed a majority of substitutions, and deletes respectively when examining single errors. Unfortunately most publications do not provide sufficient information or source errors to allow revised calculations to be made. As we describe in Chapter 5, our implementation of the model, produces a broad range of metrics including single, double, complex, mixed, and single-type errors. We can also derive these values from raw patient data, so access to the original data used for historic publications may provide more information. We also examined another possible functional relationship between both disorders, namely the steepness of the word-length curve. Data from seventeen patients, showed a significant effect, yet more data from more patients can only serve to refine the nature of the relationship between the steepness of the word length effect, and a possible indication of where the locus of impairment may lie.

Thirdly, we investigated the nature of CV orthography on error rates. Theoretical *tiers* associated with a word's constituent features (e.g. letter identity, consonant/vowel status, syllable identity, and geminate status) have often provided different rationales behind error behaviour (e.g. Caramazza & Miceli, 1990, Kay & Hanley, 1994, Jonsdottir et al, 1996, Buchwald & Rapp, 2003, Buchwald & Rapp, 2006). As the model contains no ostensive phonological mechanism, a difference in error rates between simple and complex CV words can only be attributed to orthography. There was a clear difference between error rates, with simple CV words being less prone

to error than complex CV words. Interestingly, when damage was applied to network B (the semantic system), there was still an effect albeit less substantial, thus suggesting (at least in the model) that a functional relationship may also exist with the CV effect. In other words, damage to the GOB shows a more pronounced difference between the error rates of simple and complex CV words, than when damage is applied indirectly through suboptimal input to the GOB.

Lastly there have been references to a minimum complexity principle with respect to the structure of erroneous words (e.g. Caramazza & Miceli, 1990, Kay & Hanley, 1994, Glasspool 1998). By defining a quotient we were able to compare the orthographic complexity of erroneous words to their target values. The model almost always produced erroneous words, with more simple orthographies. For two patients where we had access to all their raw data (AS, and BA), the effect was also very noticeable. It could be argued, that since the model showed fewer errors on simple than complex CV words, that there may be an implicit interrelationship between CV pairs in the corpus. This may be typical of an Italian corpus, but our corpus was composed of English words. Furthermore, English speaking patients AS and BA, who did not produce more errors on simple than complex CV words, also showed a consistent simplification of orthographic structure in the presence of error. In comparing the performance of the model to that of patients, the model actually showed a smaller decrease in complexity than AS and BA.

In summary, the original model defined by Glasspool (1998) based, to various degrees on Houghton et al (1994) and Shallice et al (1995) was extended further by Glasspool & Houghton (2005), Glasspool et al (2006) and us. The role of the graphemic buffer is to maintain the activation level of orthographic strings generated by the lexical and/or non-lexical processes while sequential letter production takes place. Clearly this

process is more difficult than it might appear to be at a superficial level. Our extended investigations (Chapters 6 and 7) highlighted a number of new behavioural aspects of the model that are also evident in some patient behaviour. These include 1) conformance of repair strategies to a minimum complexity principle, 2) different error rates for simple versus complex CV words, and 3) the existence of *pseudo-geminates*, or geminates that may not necessarily result from an active geminate status. Our investigations in Chapter 7 to explaining geminates showed mixed results, but we believe that our model is open to further development, and resolves some of the limitations discussed by Glasspool & Houghton (2005).

Cipolotti et al (2004) investigated the possibility that GBD and DD are related putative functional syndromes. Given the absence of adequate data in the literature, there are plainly some issues to be overcome in terms of substantiating these claims without further evidence. However, their classifications seem logical, and we believe that we have made some progress in supporting their claim. Indeed, the model supports the behaviour of a potential new type A and B characteristic for describing the behaviour of world-length curves; a significant difference in word-length steepness.

There is plainly ample scope for developing our model further. We would argue however, that our systematic approach to testing and analysing the model and its results has allowed us to critically analyse the model in more detail than previously.

# Appendix A General Network Parameters

| Description | Symbol | Value |
|---|---|---|
| Inhibition scaling factor | $f$ | 0.05 |
| Training Momentum | | 0.90 |
| Learning Rate | | 0.001 |
| Over-learning margin (for all thresholds) | | 0.025 |
| IE curve steepness decay parameter | F | 0.60 |
| Letter threshold (a valid output) | $T_r$ | 0.80 |
| Stop Threshold (cease recall) | $T_s$ | 0.60 |
| Geminate presentation threshold | $T_G$ | 0.80 |
| Non-Geminate threshold (target value) | $T_N$ | |
| Damping value used to inhibit geminate status | $d$ | 0.85 |
| Non Geminate threshold increase | | 0.15 |

# Appendix B Describing Error Behaviour

Throughout the thesis we suggest that representing error behaviour in a consistent fashion may be difficult especially when natural language is used to describe that behaviour. For Example, "*the behaviour of the error over serial position follows a bow shaped curve*" might describe a typical serial effect over a five-letter word. This seems quite specific, but was the curve deep, and consistent in height? Was the bow-shaped curve like a U, or like an upside-down U? Was it rounded, or pointed? Did it seem normally distributed, or was it skewed somewhat, and if so, in which direction? In this section, we describe here a minimal vocabulary, or descriptive representation that attempts to represent such behaviours. We will also review an alternative approach to describing serial position errors based roughly on the oft-cited Wing & Baddeley (1980) approach. The Wing & Baddeley approach can lead to critique around the data used to describe a patient's behaviour. Caramazza & Miceli (1990), for example, analysed results using six letter words thereby overtly biasing the importance of medial errors in position three. This section provides some techniques for describing serial position behaviour, and summarise an approach by Shallice (personal communication) for providing a more balanced accounting of errors across serial positions.

## *B.1. Serial Position Behaviour*

Serial position behaviour can be described in many ways including the curve shape and where most errors reside. This representation uses four characters describing the most distinctive attributes of a serial position curve. There was scope for adding even more attributes, but the seminal features seem for the most part to be addressed by these four:

| Overall Shape | Slant / Gradient | Inflection | Skew |
|---|---|---|---|

The following tables describe in further detail the behaviour represented by each of these character attributes. Some examples of these can be found in Figure B.1.

### B.1.1 Overall Shape of behaviour

This character describes the overall shape of the serial position curve and is a general description of the shape, further refined by the following three characters.

| Character | Overall Shape |
|---|---|
| – | Horizontal – no clear slope up or down |
| \ | Sloping downwards |
| / | Sloping upwards |
| Λ | Peaking in a medial position (Inverted U) |
| U | Initial peak degrading to a medial low and back up again |
| M | Rapid increase, rapid decrease, rapid increase and rapid decrease |
| W | Rapid decrease, rapid increase, rapid decrease and rapid increase |
| N | Increase, followed by decrease, followed by increase |
| И | Decrease from peak, increase to peak and then decrease again |

**Table B.1: Icons describing the overall shape of a serial error curve.**

### B.1.2 Slant / Gradient

This character describes whether the shape seems to slant up, down, or shows no slant. In the case where the serial position curve is complex (e.g. Λ, ,И M, N), this would represent a the nature of a trend line, say from least-squares regression.

| Character | Shape Consistency |
|---|---|
| \ | Slants down |
| / | Slants up |
| – | No Slant |

**Table B.2: Icons describing the consistency of the serial error curve.**

### B.1.3 Inflection

Most shapes should show inflection points. The N shape for example, produces two inflection points, possibly in positions two and four. With an inverted U, one might

expect the inflection point in the middle (position three). This character describes whether the inflection is as expected, or to the left or right of where expected.

| Character | Positional skew |
|---|---|
| < | Inflection is to the left of where expected. |
| > | Inflection is to the right of where expected. |
| – | Inflection is where expected. |

**Table B.3: Icons describing the positional skew of the serial error curve.**

The following table describes our expected inflection points

| Character | Expected Inflection Points |
|---|---|
| – | No inflection – should show '–'. |
| \ | No inflection – should show '–'. |
| / | No inflection – should show '–'. |
| Λ | Should inflect in position three. |
| U | Should inflect in position three. |
| M | Should inflect in position two, three, and four. |
| W | Should inflect in position two, three, and four. |
| N | Should inflect in position two, and four. |
| И | Should inflect in position two, and four. |

**Table B.4: Expected points of inflection for each serial position curve shape.**

### B.1.4 Skew

This character describes where most of the errors reside across all five serial positions.

| Character | Depth |
|---|---|
| < | Most of the errors occur in positions one and two. |
| > | Most of the errors occur in positions four and five. |
| – | Errors are spread evenly on both halves of the word. |

**Table B.5: Icons describing the depth of the output related to the serial error curve.**

### B.1.5 Example Outputs

Figure B.1 shows some example serial output graphs and their associated representations.

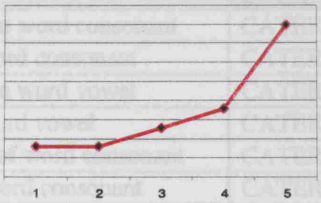
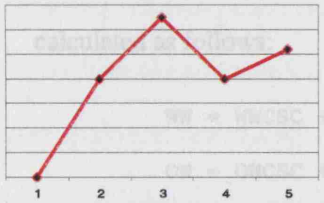
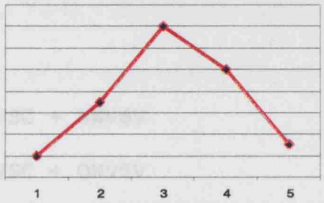
| Graph | Code | Graph | Code |
|---|---|---|---|
|  | Λ\-< |  | //-> |
|  | И\-< |  | M\-< |
|  | N/>> |  | Λ--> |

**Figure B.1: Sample graphical outputs and typical textual short-codes**

## *B.2. Error calculations*

In chapter 6, we refer to the ratios of specific error types associated with various lesion locations and severities. As well as showing specific distributions of substitutions, inserts, transpositions, and deletes, we also clarify the nature of each. For example, are substitutions within or out of class, and do they involve letters from the target word?

### B.2.1 Substitution Types

The literature shows a prevalence of within-class substitutions for example within word, and within consonant/vowel groupings (e.g. Caramazza et al 1987, Caramazza & Miceli, 1990, Cubelli, 1991). In our analyses, we collate eight different substitution types into four composite groupings so as to allow a broader comparative analysis:

| Type | Substitution Type | Example |
|---|---|---|
| WWCSC | Consonant substituted by a **within** word consonant | CATER → CA**R**ER |
| WWCSV | Vowel substituted by a **within** word consonant | CATER → CAT**C**R |
| WWVSC | Consonant substituted by a **within** word vowel | CATER → **E**ATER |
| WWVSV | Vowel substituted by a **within** word vowel | CATER → CAT**A**R |
| OWCSC | Consonant substituted by an **out of** word consonant | CATER → **W**ATER |
| OWCSV | Vowel substituted by an **out of** word consonant | CATER → CAT**P**R |
| OWVSC | Consonant substituted by an **out of** word vowel | CATER → CA**O**ER |
| OWVSV | Vowel substituted by an **out of** word vowel | CATER → CAT**U**R |

**Table B.6 : Description of substitution types being analysed**

1. **Within-word substitutions (WW) compared to Out-of-word (OW) substitutions.**

   These indicate how much a letter's identity is tied to the word it belongs to, and are calculated as follows:

   $$WW = WWCSC + WWCSV + WWVSC + WWVSV \quad \textbf{Equation B.1}$$

   $$OW = OWCSC + OWCSV + OWVSC + OWVSV \quad \textbf{Equation B.2}$$

2. **Substitutions by consonants (CON) compared to by vowels (VOW).**

   These indicate whether substitutions are more likely to occur by a consonant or vowel (irrespective of within or out-of word) and are calculated as follows:

   $$CON = WWCSC + WWCSV + OWCSC + OWCSV \quad \textbf{Equation B.3}$$

   $$VOW = WWVSC + WWVSV + OWVSC + OWVSV \quad \textbf{Equation B.4}$$

3. **Substitutions within CV class (SAME) compared to across CV class (DIFF).**

   These indicate how much effect consonant/vowel status has on substitutions and are calculated as follows:

   $$SAME = WWCSC + WWVSV + OWCSC + OWVSV \quad \textbf{Equation B.5}$$

   $$DIFF = WWCSV + WWVSC + OWCSV + OWVSC \quad \textbf{Equation B.6}$$

4. **Overall distribution of errors.**

   These indicate the distribution of error types (irrespective of within/out-of word) and are calculated as follows:

$$CSC = WWCSC + OWCSC \quad \textbf{Equation B.7}$$

$$CSV = WWCSV + OWCSV \quad \textbf{Equation B.8}$$

$$VSC = WWVSC + OWVSC \quad \textbf{Equation B.9}$$

$$VSV = WWVSV + OWVSV \quad \textbf{Equation B.10}$$

### B.2.2 Transposition Types

*There are various approaches to classifying transpositions. Caramazza & Miceli (1990) and Tainturier & Caramazza (1996) for example differentiate between shifts and exchanges, whereas others (e.g. Sage & Ellis, 2004), consider any obvious movement of a letter from one position in a word to another as a transposition. We have chosen the latter approach, and classify them as follows:*

| Type | Transposition Type | Example |
|---|---|---|
| CC | Consonant Transposed with Consonant | CATER → TACER |
| CV | Consonant Transposed with Vowel | CATER → ACTER |
| VV | Vowel Transposed with Vowel | CATER → CETAR |

**Table B.7 : Description of transposition types being analysed**

1. **Transpositions within CV class (SAME) compared to across (DIFF) class**

   This gives an indication of how significantly transpositions take place within class and is calculated as follows:

$$SAME = CC + VV \quad \textbf{Equation B.11}$$

$$DIFF = CV \quad \textbf{Equation B.12}$$

## *B.3 Measurement of Positional Errors*

Wing and Baddeley (1980) describe a method of normalising errors across five serial positions thus providing a common serial representation across multiple word lengths. Their basic mechanism outlined in Chapter 5 has some inherent problems, which we believe can be remedied by an approach providing a more balanced accountability across

five serial positions (Shallice, personal communication). Unlike the Wing & Baddeley (WB) method, the modified approach allows errors to contribute partial values across a number of serial positions. We should expect that serial position curves in the literature, and our new *interpretation* of the same data to be qualitatively similar, and this indeed seems to be the case. Figure B.2 for example, compares the normalised curves for six letter words for patient LB (Caramazza & Miceli, 1990) using the WB and new approaches. With the WB method, six letter words tend to accentuate the peak of the curve by counting errors in positions three and four as belong to normalised position three. In the new approach, these errors are more fairly shared across positions two, three, and four.

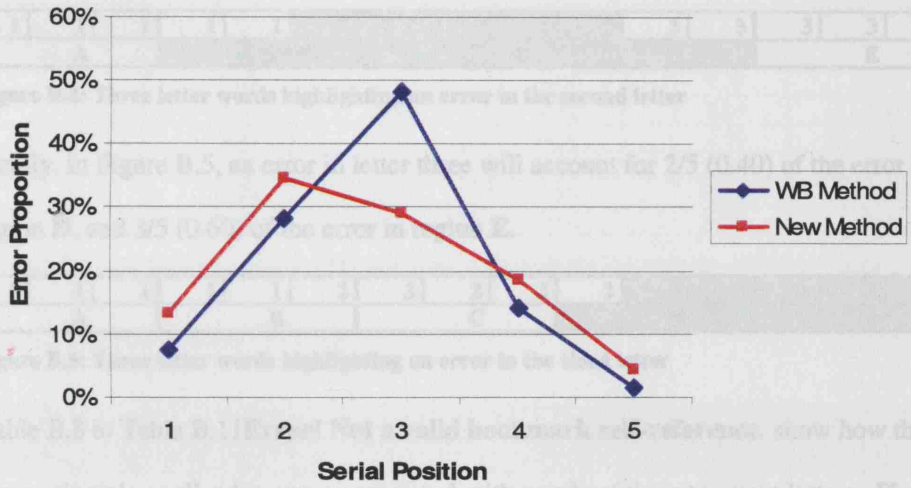



**Figure B.2: A comparison of serial error positions calculated using the WB and new method**

### B.3.1 Serial Position Accountability

The following examples (Figure B.3, Figure B.4, and Figure B.5) demonstrate how we account for three letter words spread across five serial positions. Each letter position (A-E) is subdivided into the number of serial positions for the word being analysed. An error is then accounted for, by summing the proportion of errors for each serial position.

### B.3.1.1 Three Letter Words

Figure B.3 for example, shows that an error in letter one (shaded in grey) is accountable in part to both regions **A** and **B** (shaded in green). The extent of the accountability is calculated by counting the number of segments in the top row matching the associated region. Therefore, an error in position one will have 3/5 (0.60) of the entire error in region **A**, and 2/5 (0.40) of the entire error in region **B**.

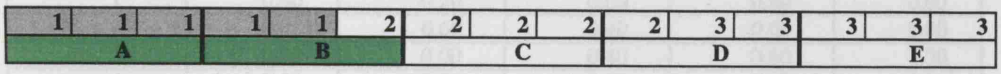



**Figure B.3: Three letter words highlighting an error in the first letter**

Similarly, in Figure B.4, an error in letter two will account for 1/5 (0.20) of the error in region **B**, 3/5 (0.60) of the error in region **C**, and 1/5 (0.20) of the error in region **D**.

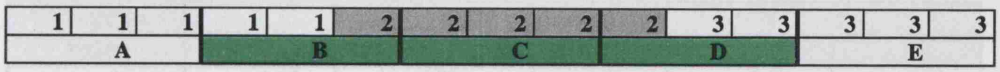



**Figure B.4: Three letter words highlighting an error in the second letter**

Finally, in Figure B.5, an error in letter three will account for 2/5 (0.40) of the error in region **D**, and 3/5 (0.60) of the error in region **E**.

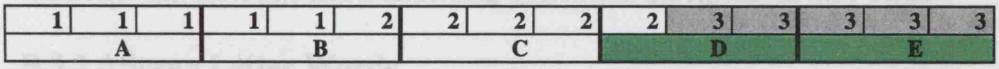



**Figure B.5: Three letter words highlighting an error in the third letter**

Table B.8 to Table B.11**Error! Not a valid bookmark self-reference.** show how the same principle applies for errors associated with words of three to seven letters. Five-letter words will have a one to one correspondence between region and serial position.

| Error in letter | Region | | | | |
|---|---|---|---|---|---|
| | A | B | C | D | E |
| 1 | 0.60 | 0.40 | 0.00 | 0.00 | 0.00 |
| 2 | 0.00 | 0.20 | 0.60 | 0.20 | 0.00 |
| 3 | 0.00 | 0.00 | 0.00 | 0.40 | 0.60 |

**Table B.8: Alternative approach to distributing errors across five regions– three letter words.**

### B.3.1.2 Four Letter Words

| Error in letter | Region | | | | |
|---|---|---|---|---|---|
| | A | B | C | D | E |
| 1 | 0.80 | 0.20 | 0.00 | 0.00 | 0.00 |
| 2 | 0.00 | 0.60 | 0.40 | 0.00 | 0.00 |
| 3 | 0.00 | 0.00 | 0.40 | 0.60 | 0.00 |
| 4 | 0.00 | 0.00 | 0.00 | 0.20 | 0.80 |

**Table B.9: Alternative approach to distributing errors across five regions – four letter words.**

### B.3.1.3 Six Letter Words

| Error in letter | Region | | | | |
|---|---|---|---|---|---|
| | A | B | C | D | E |
| 1 | 1.00 | 0.00 | 0.00 | 0.00 | 0.00 |
| 2 | 0.20 | 0.80 | 0.00 | 0.00 | 0.00 |
| 3 | 0.00 | 0.40 | 0.60 | 0.00 | 0.00 |
| 4 | 0.00 | 0.00 | 0.60 | 0.40 | 0.00 |
| 5 | 0.00 | 0.00 | 0.00 | 0.80 | 0.20 |
| 6 | 0.00 | 0.00 | 0.00 | 0.00 | 1.00 |

**Table B.10: Alternative approach to distributing errors across five regions– six letter words.**

### B.3.1.4 Seven Letter Words

| Error in letter | Position | | | | |
|---|---|---|---|---|---|
| | A | B | C | D | E |
| 1 | 1.00 | 0.00 | 0.00 | 0.00 | 0.00 |
| 2 | 0.40 | 0.60 | 0.00 | 0.00 | 0.00 |
| 3 | 0.00 | 0.80 | 0.20 | 0.00 | 0.00 |
| 4 | 0.00 | 0.00 | 1.00 | 0.00 | 0.00 |
| 5 | 0.00 | 0.00 | 0.20 | 0.80 | 0.00 |
| 6 | 0.00 | 0.00 | 0.00 | 0.60 | 0.40 |
| 7 | 0.00 | 0.00 | 0.00 | 0.00 | 1.00 |

**Table B.11: Alternative approach to distributing errors across five positions – seven letter words.**

# Appendix C Geminate Corpus

The new corpus testing geminate related symptoms based on the corpus by Ward (1998).

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| ARAB | GERM | SKIN | CIDER | MELON | TWEED | HORROR | VISION |
| PITA | GULF | SPIT | CLOAK | MODEL | VAGUE | INFANT | WISDOM |
| PALL | GROW | SWAN | CRIMP | NASTY | VOICE | JERSEY | ABDOMEN |
| BASE | HATE | TAPE | CRANE | NOISE | WHARF | LADDER | BAYONET |
| BEER | HIDE | THIN | CROSS | OLIVE | ZEBRA | MAMMAL | BITCHER |
| BEND | HOLD | TOSS | DANCE | PANDA | ADVICE | MENACE | CENTURY |
| BLOW | HIND | STIR | DOZEN | PERCY | ASLEEP | MITTEN | COMRADE |
| BONE | ITEM | UNIT | DRILL | POUCH | AUTUMN | MURDER | COTTAGE |
| YOKE | JOKE | VERB | EBONY | PRUNE | CRAVER | NARROW | DIPLOMA |
| CALM | KEEP | GRAM | ESSAY | QUIET | BORDER | NEPHEW | EPISODE |
| CHEF | KNEE | WINE | FALSE | REBEL | BRIGHT | PALACE | FRANCIS |
| CLAY | LAND | ABBOT | FASTS | RINSE | MUTTER | PILLOW | GIRAFFE |
| COMB | LAZY | AGONY | FEVER | ROYAL | CASTLE | POCKET | JOURNAL |
| COOK | CRUD | ALLOY | FLAME | SCENE | CENTRE | PUZZLE | KNUCKLE |
| CROW | GUSH | ANKLE | FOCUS | SHALE | CINEMA | CATTLE | MAJESTY |
| CART | SOOT | AROMA | GHOST | SHOOK | COWARD | REGION | NURSERY |
| BITE | CIGS | AVERT | GLOVE | SINGE | DANGER | RIBBON | PATTERN |
| DOLL | OATH | BASIS | CRASS | SLICE | DEPUTY | SALOON | RECEIPT |
| CULL | PAWN | WINCH | HODGE | SPEAK | DOLLAR | SCHOOL | ROOSTER |
| ECHO | PEST | BRAIN | HOUND | STAFF | ENERGY | SHIELD | SOLDIER |
| EXIT | PREY | BROOK | IMAGE | STORE | EXPORT | SQUARE | SPECIAL |
| HARM | CENT | BUNCH | JUDGE | STING | FAMOUS | STRIKE | SUICIDE |
| TINE | RIOT | CANAL | LABEL | TIGER | FUMBLE | SYSTEM | THEATRE |
| FOLK | SAVE | CHAOS | LATHE | TRADE | GENTLE | THEORY | TROUBLE |
| FROG | GATS | CHORE | LIMIT | TRITE | HANDLE | TRAVEL | BASSOON |

# Appendix D Lesion Severities

## D.1 Network A - Geminate related lesions

| | | Lesion Type | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Lesion Severity | Lesion Location | Ablate | Noise | | Constrain | | Scale | Lesion Location | Ablate | Noise | | Constrain | | Scale |
| | | % | Lower | Upper | Lower | Upper | | | % | Lower | Upper | Lower | Upper | |
| 1 | Randomly through the network | 0.000175 | -0.0100 | 0.0100 | 0.9500 | 0.9800 | 0.9700 | Input layer (Semantic connections) | 0.0060 | -0.0025 | 0.0025 | 0.960 | 0.980 | 0.97 |
| 2 | | 0.000350 | -0.0200 | 0.0200 | 0.9425 | 0.9770 | 0.9640 | | 0.0120 | -0.0050 | 0.0050 | 0.930 | 0.965 | 0.95 |
| 3 | | 0.000525 | -0.0300 | 0.0300 | 0.9350 | 0.9740 | 0.9580 | | 0.0180 | -0.0075 | 0.0075 | 0.900 | 0.950 | 0.93 |
| 4 | | 0.000700 | -0.0400 | 0.0400 | 0.9275 | 0.9710 | 0.9520 | | 0.0240 | -0.0100 | 0.0100 | 0.870 | 0.935 | 0.91 |
| 5 | | 0.000875 | -0.0500 | 0.0500 | 0.9200 | 0.9680 | 0.9460 | | 0.0300 | -0.0125 | 0.0125 | 0.840 | 0.920 | 0.89 |
| 6 | | 0.001050 | -0.0600 | 0.0600 | 0.9125 | 0.9650 | 0.9400 | | 0.0360 | -0.0150 | 0.0150 | 0.810 | 0.905 | 0.87 |
| 7 | | 0.001225 | -0.0700 | 0.0700 | 0.9050 | 0.9620 | 0.9340 | | 0.0420 | -0.0175 | 0.0175 | 0.780 | 0.890 | 0.85 |
| 8 | | 0.001400 | -0.0800 | 0.0800 | 0.8975 | 0.9590 | 0.9280 | | 0.0480 | -0.0200 | 0.0200 | 0.750 | 0.875 | 0.83 |
| 9 | | 0.001575 | -0.0900 | 0.0900 | 0.8900 | 0.9560 | 0.9220 | | 0.0540 | -0.0225 | 0.0225 | 0.720 | 0.860 | 0.81 |
| 10 | | 0.001750 | -0.1000 | 0.1000 | 0.8825 | 0.9530 | 0.9160 | | 0.0600 | -0.0250 | 0.0250 | 0.690 | 0.845 | 0.79 |
| 1 | Input Layer (all connections) | 0.00125 | -0.0165 | 0.0165 | 0.9750 | 1.0000 | 0.9800 | Hidden Layer connections | 0.00150 | -0.0200 | 0.0200 | 0.9650 | 0.9800 | 0.9000 |
| 2 | | 0.00250 | -0.0330 | 0.0330 | 0.9560 | 0.9925 | 0.9660 | | 0.00300 | -0.0325 | 0.0325 | 0.9475 | 0.9725 | 0.8950 |
| 3 | | 0.00375 | -0.0495 | 0.0495 | 0.9370 | 0.9850 | 0.9520 | | 0.00450 | -0.0450 | 0.0450 | 0.9300 | 0.9650 | 0.8900 |
| 4 | | 0.00500 | -0.0660 | 0.0660 | 0.9180 | 0.9775 | 0.9380 | | 0.00600 | -0.0575 | 0.0575 | 0.9125 | 0.9575 | 0.8850 |
| 5 | | 0.00625 | -0.0825 | 0.0825 | 0.8990 | 0.9700 | 0.9240 | | 0.00750 | -0.0700 | 0.0700 | 0.8950 | 0.9500 | 0.8800 |
| 6 | | 0.00750 | -0.0990 | 0.0990 | 0.8800 | 0.9625 | 0.9100 | | 0.00900 | -0.0825 | 0.0825 | 0.8775 | 0.9425 | 0.8750 |
| 7 | | 0.00875 | -0.1155 | 0.1155 | 0.8610 | 0.9550 | 0.8960 | | 0.01050 | -0.0950 | 0.0950 | 0.8600 | 0.9350 | 0.8700 |
| 8 | | 0.01000 | -0.1320 | 0.1320 | 0.8420 | 0.9475 | 0.8820 | | 0.01200 | -0.1075 | 0.1075 | 0.8425 | 0.9275 | 0.8650 |
| 9 | | 0.01125 | -0.1485 | 0.1485 | 0.8230 | 0.9400 | 0.8680 | | 0.01350 | -0.1200 | 0.1200 | 0.8250 | 0.9200 | 0.8600 |
| 10 | | 0.01250 | -0.1650 | 0.1650 | 0.8040 | 0.9325 | 0.8540 | | 0.01500 | -0.1325 | 0.1325 | 0.8075 | 0.9125 | 0.8550 |
| 1 | Input Layer (Context Node connections) | 0.0020 | -0.0200 | 0.0200 | 0.970 | 1.000 | 0.9750 | Output Layer | N/A | -0.0050 | 0.0050 | N/A | | |
| 2 | | 0.0040 | -0.0400 | 0.0400 | 0.945 | 0.991 | 0.9585 | | | -0.0100 | 0.0100 | | | |
| 3 | | 0.0060 | -0.0600 | 0.0600 | 0.920 | 0.982 | 0.9420 | | | -0.0150 | 0.0150 | | | |
| 4 | | 0.0080 | -0.0800 | 0.0800 | 0.895 | 0.973 | 0.9255 | | | -0.0200 | 0.0200 | | | |
| 5 | | 0.0100 | -0.1000 | 0.1000 | 0.870 | 0.964 | 0.9090 | | | -0.0250 | 0.0250 | | | |
| 6 | | 0.0120 | -0.1200 | 0.1200 | 0.845 | 0.955 | 0.8925 | | | -0.0300 | 0.0300 | | | |
| 7 | | 0.0140 | -0.1400 | 0.1400 | 0.820 | 0.946 | 0.8760 | | | -0.0350 | 0.0350 | | | |
| 8 | | 0.0160 | -0.1600 | 0.1600 | 0.795 | 0.937 | 0.8595 | | | -0.0400 | 0.0400 | | | |
| 9 | | 0.0180 | -0.1800 | 0.1800 | 0.770 | 0.928 | 0.8430 | | | -0.0450 | 0.0450 | | | |
| 10 | | 0.0200 | -0.2000 | 0.2000 | 0.745 | 0.919 | 0.8265 | | | -0.0500 | 0.0500 | | | |

## D.2 Network A – Moving Window Context

| Lesion Severity | Lesion Location | Lesion Type: Ablate % | Noise: Lower | Noise: Upper | Constrain: Lower | Constrain: Upper | Scale | Lesion Location | Lesion Type: Ablate % | Noise: Lower | Noise: Upper | Constrain: Lower | Constrain: Upper | Scale |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Randomly through the network | 0.000025 | -0.0175 | 0.0175 | 0.9500 | 0.9800 | 0.9800 | Input layer (Semantic connections) | 0.0010 | -0.0500 | 0.0500 | 0.960 | 0.98 | 0.97 |
| 2 | | 0.000150 | -0.0350 | 0.0350 | 0.9400 | 0.9750 | 0.9700 | | 0.0020 | -0.1000 | 0.1000 | 0.920 | 0.96 | 0.94 |
| 3 | | 0.000275 | -0.0525 | 0.0525 | 0.9300 | 0.9700 | 0.9600 | | 0.0030 | -0.1500 | 0.1500 | 0.880 | 0.94 | 0.91 |
| 4 | | 0.000400 | -0.0700 | 0.0700 | 0.9200 | 0.9650 | 0.9500 | | 0.0040 | -0.2000 | 0.2000 | 0.840 | 0.92 | 0.88 |
| 5 | | 0.000525 | -0.0875 | 0.0875 | 0.9100 | 0.9600 | 0.9400 | | 0.0050 | -0.2500 | 0.2500 | 0.800 | 0.90 | 0.85 |
| 6 | | 0.000650 | -0.1050 | 0.1050 | 0.9000 | 0.9550 | 0.9300 | | 0.0060 | -0.3000 | 0.3000 | 0.760 | 0.88 | 0.82 |
| 7 | | 0.000775 | -0.1225 | 0.1225 | 0.8900 | 0.9500 | 0.9200 | | 0.0070 | -0.3500 | 0.3500 | 0.720 | 0.86 | 0.79 |
| 8 | | 0.000900 | -0.1400 | 0.1400 | 0.8800 | 0.9450 | 0.9100 | | 0.0080 | -0.4000 | 0.4000 | 0.680 | 0.84 | 0.76 |
| 9 | | 0.001025 | -0.1575 | 0.1575 | 0.8700 | 0.9400 | 0.9000 | | 0.0090 | -0.4500 | 0.4500 | 0.640 | 0.82 | 0.73 |
| 10 | | 0.001150 | -0.1750 | 0.1750 | 0.8600 | 0.9350 | 0.8900 | | 0.0100 | -0.5000 | 0.5000 | 0.600 | 0.80 | 0.70 |
| 1 | Input Layer (all connections) | 0.0015 | -0.0280 | 0.0280 | 0.975 | 1.00 | 0.98 | Hidden Layer connections | 0.00150 | -0.0200 | 0.0200 | 0.975 | 0.99 | 0.8700 |
| 2 | | 0.0030 | -0.0560 | 0.0560 | 0.950 | 0.99 | 0.96 | | 0.00300 | -0.0400 | 0.0400 | 0.950 | 0.98 | 0.8650 |
| 3 | | 0.0045 | -0.0840 | 0.0840 | 0.925 | 0.98 | 0.94 | | 0.00450 | -0.0600 | 0.0600 | 0.925 | 0.97 | 0.8600 |
| 4 | | 0.0060 | -0.1120 | 0.1120 | 0.900 | 0.97 | 0.92 | | 0.00600 | -0.0800 | 0.0800 | 0.900 | 0.96 | 0.8550 |
| 5 | | 0.0075 | -0.1400 | 0.1400 | 0.875 | 0.96 | 0.90 | | 0.00750 | -0.1000 | 0.1000 | 0.875 | 0.95 | 0.8500 |
| 6 | | 0.0090 | -0.1680 | 0.1680 | 0.850 | 0.95 | 0.88 | | 0.00900 | -0.1200 | 0.1200 | 0.850 | 0.94 | 0.8450 |
| 7 | | 0.0105 | -0.1960 | 0.1960 | 0.825 | 0.94 | 0.86 | | 0.01050 | -0.1400 | 0.1400 | 0.825 | 0.93 | 0.8400 |
| 8 | | 0.0120 | -0.2240 | 0.2240 | 0.800 | 0.93 | 0.84 | | 0.01200 | -0.1600 | 0.1600 | 0.800 | 0.92 | 0.8350 |
| 9 | | 0.0135 | -0.2520 | 0.2520 | 0.775 | 0.92 | 0.82 | | 0.01350 | -0.1800 | 0.1800 | 0.775 | 0.91 | 0.8300 |
| 10 | | 0.0150 | -0.2800 | 0.2800 | 0.750 | 0.91 | 0.80 | | 0.01500 | -0.2000 | 0.2000 | 0.750 | 0.90 | 0.8250 |
| 1 | Input Layer (Context Node connections) | 0.0020 | -0.0300 | 0.0300 | 0.970 | 1.00 | 0.975 | Output Layer | N/A | -0.0050 | 0.0050 | N/A | | |
| 2 | | 0.0040 | -0.0600 | 0.0600 | 0.940 | 0.99 | 0.950 | | | -0.0100 | 0.0100 | | | |
| 3 | | 0.0060 | -0.0900 | 0.0900 | 0.910 | 0.98 | 0.925 | | | -0.0150 | 0.0150 | | | |
| 4 | | 0.0080 | -0.1200 | 0.1200 | 0.880 | 0.97 | 0.900 | | | -0.0200 | 0.0200 | | | |
| 5 | | 0.0100 | -0.1500 | 0.1500 | 0.850 | 0.96 | 0.875 | | | -0.0250 | 0.0250 | | | |
| 6 | | 0.0120 | -0.1800 | 0.1800 | 0.820 | 0.95 | 0.850 | | | -0.0300 | 0.0300 | | | |
| 7 | | 0.0140 | -0.2100 | 0.2100 | 0.790 | 0.94 | 0.825 | | | -0.0350 | 0.0350 | | | |
| 8 | | 0.0160 | -0.2400 | 0.2400 | 0.760 | 0.93 | 0.800 | | | -0.0400 | 0.0400 | | | |
| 9 | | 0.0180 | -0.2700 | 0.2700 | 0.730 | 0.92 | 0.775 | | | -0.0450 | 0.0450 | | | |
| 10 | | 0.0200 | -0.3000 | 0.3000 | 0.700 | 0.91 | 0.750 | | | -0.0500 | 0.0500 | | | |

## al Context

| Lesion Type | | | | | | Lesion Location | Lesion Type | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Noise | | Constrain | | Scale | | Ablate | Noise | | Constrain | | Scale |
| | Lower | Upper | Lower | Upper | | | % | Lower | Upper | Lower | Upper | |
| 0040 | -0.030 | 0.030 | 0.942 | 0.985 | 0.9600 | Input layer (Semantic connections) | 0.0003 | -0.04 | 0.04 | 0.92 | 0.960 | 0.9300 |
| 0125 | -0.045 | 0.045 | 0.934 | 0.980 | 0.9545 | | 0.0006 | -0.08 | 0.08 | 0.90 | 0.951 | 0.9175 |
| 0210 | -0.060 | 0.060 | 0.926 | 0.975 | 0.9490 | | 0.0009 | -0.12 | 0.12 | 0.88 | 0.942 | 0.9050 |
| 0295 | -0.075 | 0.075 | 0.918 | 0.970 | 0.9435 | | 0.0012 | -0.16 | 0.16 | 0.86 | 0.933 | 0.8925 |
| 0380 | -0.090 | 0.090 | 0.910 | 0.965 | 0.9380 | | 0.0015 | -0.20 | 0.20 | 0.84 | 0.924 | 0.8800 |
| 0465 | -0.105 | 0.105 | 0.902 | 0.960 | 0.9325 | | 0.0018 | -0.24 | 0.24 | 0.82 | 0.915 | 0.8675 |
| 0550 | -0.120 | 0.120 | 0.894 | 0.955 | 0.9270 | | 0.0021 | -0.28 | 0.28 | 0.80 | 0.906 | 0.8550 |
| 0635 | -0.135 | 0.135 | 0.886 | 0.950 | 0.9215 | | 0.0024 | -0.32 | 0.32 | 0.78 | 0.897 | 0.8425 |
| 0720 | -0.150 | 0.150 | 0.878 | 0.945 | 0.9160 | | 0.0027 | -0.36 | 0.36 | 0.76 | 0.888 | 0.8300 |
| 0805 | -0.165 | 0.165 | 0.870 | 0.940 | 0.9105 | | 0.0030 | -0.40 | 0.40 | 0.74 | 0.879 | 0.8175 |
| 0010 | -0.050 | 0.050 | 0.920 | 0.980 | 0.94 | Hidden Layer connections | 0.001 | -0.02 | 0.02 | 0.975 | 0.99 | 0.89 |
| 0035 | -0.085 | 0.085 | 0.905 | 0.974 | 0.93 | | 0.002 | -0.04 | 0.04 | 0.950 | 0.98 | 0.88 |
| 0060 | -0.120 | 0.120 | 0.890 | 0.968 | 0.92 | | 0.003 | -0.06 | 0.06 | 0.925 | 0.97 | 0.87 |
| 0085 | -0.155 | 0.155 | 0.875 | 0.962 | 0.91 | | 0.004 | -0.08 | 0.08 | 0.900 | 0.96 | 0.86 |
| 0110 | -0.190 | 0.190 | 0.860 | 0.956 | 0.90 | | 0.005 | -0.10 | 0.10 | 0.875 | 0.95 | 0.85 |
| 0135 | -0.225 | 0.225 | 0.845 | 0.950 | 0.89 | | 0.006 | -0.12 | 0.12 | 0.850 | 0.94 | 0.84 |
| 0160 | -0.260 | 0.260 | 0.830 | 0.944 | 0.88 | | 0.007 | -0.14 | 0.14 | 0.825 | 0.93 | 0.83 |
| 0185 | -0.295 | 0.295 | 0.815 | 0.938 | 0.87 | | 0.008 | -0.16 | 0.16 | 0.800 | 0.92 | 0.82 |
| 0210 | -0.330 | 0.330 | 0.800 | 0.932 | 0.86 | | 0.009 | -0.18 | 0.18 | 0.775 | 0.91 | 0.81 |
| 0235 | -0.365 | 0.365 | 0.785 | 0.926 | 0.85 | | 0.010 | -0.20 | 0.20 | 0.750 | 0.90 | 0.80 |
| 0100 | -0.15 | 0.15 | 0.97 | 1.00 | 0.975 | Output Layer | N/A | -0.0075 | 0.0075 | N/A | | |
| 0450 | -0.21 | 0.21 | 0.94 | 0.99 | 0.950 | | | -0.015 | 0.015 | | | |
| 0800 | -0.27 | 0.27 | 0.91 | 0.98 | 0.925 | | | -0.0225 | 0.0225 | | | |
| 0150 | -0.33 | 0.33 | 0.88 | 0.97 | 0.900 | | | -0.03 | 0.03 | | | |
| 0500 | -0.39 | 0.39 | 0.85 | 0.96 | 0.875 | | | -0.0375 | 0.0375 | | | |
| 0850 | -0.45 | 0.45 | 0.82 | 0.95 | 0.850 | | | -0.045 | 0.045 | | | |
| 0200 | -0.51 | 0.51 | 0.79 | 0.94 | 0.825 | | | -0.0525 | 0.0525 | | | |
| 0550 | -0.57 | 0.57 | 0.76 | 0.93 | 0.800 | | | -0.06 | 0.06 | | | |
| 0900 | -0.63 | 0.63 | 0.73 | 0.92 | 0.775 | | | -0.0675 | 0.0675 | | | |
| 0250 | -0.69 | 0.69 | 0.70 | 0.91 | 0.750 | | | -0.075 | 0.075 | | | |

## D.4 Network A - IE Context

| Lesion Severity | Lesion Location | Lesion Type: Ablate % | Noise: Lower | Noise: Upper | Constrain: Lower | Constrain: Upper | Scale |
|---|---|---|---|---|---|---|---|
| 1 | Randomly through the network | 0.000040 | -0.015 | 0.015 | 0.9900 | 1.0000 | 0.990 |
| 2 | | 0.000125 | -0.030 | 0.030 | 0.9650 | 0.9900 | 0.975 |
| 3 | | 0.000210 | -0.045 | 0.045 | 0.9400 | 0.9800 | 0.960 |
| 4 | | 0.000295 | -0.060 | 0.060 | 0.9150 | 0.9700 | 0.945 |
| 5 | | 0.000380 | -0.075 | 0.075 | 0.8900 | 0.9600 | 0.930 |
| 6 | | 0.000465 | -0.090 | 0.090 | 0.8650 | 0.9500 | 0.915 |
| 7 | | 0.000550 | -0.105 | 0.105 | 0.8400 | 0.9400 | 0.900 |
| 8 | | 0.000635 | -0.120 | 0.120 | 0.8150 | 0.9300 | 0.885 |
| 9 | | 0.000720 | -0.135 | 0.135 | 0.7900 | 0.9200 | 0.870 |
| 10 | | 0.000805 | -0.150 | 0.150 | 0.7650 | 0.9100 | 0.855 |
| 1 | Input Layer (all connections) | 0.0015 | -0.0275 | 0.0275 | 0.9750 | 1.000 | 0.98 |
| 2 | | 0.0030 | -0.0550 | 0.0550 | 0.9475 | 0.991 | 0.96 |
| 3 | | 0.0045 | -0.0825 | 0.0825 | 0.9200 | 0.982 | 0.94 |
| 4 | | 0.0060 | -0.1100 | 0.1100 | 0.8925 | 0.973 | 0.92 |
| 5 | | 0.0075 | -0.1375 | 0.1375 | 0.8650 | 0.964 | 0.90 |
| 6 | | 0.0090 | -0.1650 | 0.1650 | 0.8375 | 0.955 | 0.88 |
| 7 | | 0.0105 | -0.1925 | 0.1925 | 0.8100 | 0.946 | 0.86 |
| 8 | | 0.0120 | -0.2200 | 0.2200 | 0.7825 | 0.937 | 0.84 |
| 9 | | 0.0135 | -0.2475 | 0.2475 | 0.7550 | 0.928 | 0.82 |
| 10 | | 0.0150 | -0.2750 | 0.2750 | 0.7275 | 0.919 | 0.80 |
| 1 | Input Layer (Context Node connections) | 0.002 | -0.055 | 0.055 | 0.97 | 1.00 | 0.975 |
| 2 | | 0.004 | -0.110 | 0.110 | 0.94 | 0.99 | 0.950 |
| 3 | | 0.006 | -0.165 | 0.165 | 0.91 | 0.98 | 0.925 |
| 4 | | 0.008 | -0.220 | 0.220 | 0.88 | 0.97 | 0.900 |
| 5 | | 0.010 | -0.275 | 0.275 | 0.85 | 0.96 | 0.875 |
| 6 | | 0.012 | -0.330 | 0.330 | 0.82 | 0.95 | 0.850 |
| 7 | | 0.014 | -0.385 | 0.385 | 0.79 | 0.94 | 0.825 |
| 8 | | 0.016 | -0.440 | 0.440 | 0.76 | 0.93 | 0.800 |
| 9 | | 0.018 | -0.495 | 0.495 | 0.73 | 0.92 | 0.775 |
| 10 | | 0.020 | -0.550 | 0.550 | 0.70 | 0.91 | 0.750 |

| Lesion Location | Lesion Type: Ablate % | Noise: Lower | Noise: Upper | Constrain: Lower | Constrain: Upper | Scale |
|---|---|---|---|---|---|---|
| Input layer (Semantic connections) | 0.0025 | -0.04 | 0.04 | 0.960 | 0.980 | 0.9700 |
| | 0.0050 | -0.08 | 0.08 | 0.944 | 0.972 | 0.9555 |
| | 0.0075 | -0.12 | 0.12 | 0.928 | 0.964 | 0.9410 |
| | 0.0100 | -0.16 | 0.16 | 0.912 | 0.956 | 0.9265 |
| | 0.0125 | -0.20 | 0.20 | 0.896 | 0.948 | 0.9120 |
| | 0.0150 | -0.24 | 0.24 | 0.880 | 0.940 | 0.8975 |
| | 0.0175 | -0.28 | 0.28 | 0.864 | 0.932 | 0.8830 |
| | 0.0200 | -0.32 | 0.32 | 0.848 | 0.924 | 0.8685 |
| | 0.0225 | -0.36 | 0.36 | 0.832 | 0.916 | 0.8540 |
| | 0.0250 | -0.40 | 0.40 | 0.816 | 0.908 | 0.8395 |
| Hidden Layer connections | 0.00125 | -0.02 | 0.02 | 0.975 | 0.99 | 0.88 |
| | 0.00250 | -0.04 | 0.04 | 0.945 | 0.98 | 0.87 |
| | 0.00375 | -0.06 | 0.06 | 0.915 | 0.97 | 0.86 |
| | 0.00500 | -0.08 | 0.08 | 0.885 | 0.96 | 0.85 |
| | 0.00625 | -0.10 | 0.10 | 0.855 | 0.95 | 0.84 |
| | 0.00750 | -0.12 | 0.12 | 0.825 | 0.94 | 0.83 |
| | 0.00875 | -0.14 | 0.14 | 0.795 | 0.93 | 0.82 |
| | 0.01000 | -0.16 | 0.16 | 0.765 | 0.92 | 0.81 |
| | 0.01125 | -0.18 | 0.18 | 0.735 | 0.91 | 0.80 |
| | 0.01250 | -0.20 | 0.20 | 0.705 | 0.90 | 0.79 |

| Lesion Location | Lesion Type: Ablate % | Noise: Lower | Noise: Upper | Constrain | Range | Scale |
|---|---|---|---|---|---|---|
| Output Layer | N/A | -0.0050 | 0.0050 | IE Lesion (I & E types) | 0.05 | .0750 |
| | | -0.0095 | 0.0095 | | 0.05 | 0.150 |
| | | -0.0140 | 0.0140 | | 0.05 | 0.225 |
| | | -0.0185 | 0.0185 | | 0.05 | 0.300 |
| | | -0.0230 | 0.0230 | | 0.05 | 0.375 |
| | | -0.0275 | 0.0275 | | 0.05 | 0.450 |
| | | -0.0320 | 0.0320 | | 0.05 | 0.525 |
| | | -0.0365 | 0.0365 | | 0.05 | 0.600 |
| | | -0.0410 | 0.0410 | | 0.05 | 0.675 |
| | | -0.0455 | 0.0455 | | 0.05 | 0.750 |

## D.5 Network B – Plaut and Shallice Semantic Design

| Lesion Severity | Lesion Location | Lesion Type | | | | | |
|---|---|---|---|---|---|---|---|
| | | Ablate % | Noise | | Constrain | | Scale |
| | | | Lower | Upper | Lower | Upper | |
| 1 | Randomly through the network | 0.00250 | -0.1000 | 0.1000 | 0.8000 | 0.950 | 0.7500 |
| 2 | | 0.00500 | -0.1250 | 0.1250 | 0.7600 | 0.925 | 0.7175 |
| 3 | | 0.00750 | -0.1500 | 0.1500 | 0.7200 | 0.900 | 0.6850 |
| 4 | | 0.01000 | -0.1750 | 0.1750 | 0.6800 | 0.875 | 0.6525 |
| 5 | | 0.01250 | -0.2000 | 0.2000 | 0.6400 | 0.850 | 0.6200 |
| 6 | | 0.01500 | -0.2250 | 0.2250 | 0.6000 | 0.825 | 0.5875 |
| 7 | | 0.01750 | -0.2500 | 0.2500 | 0.5600 | 0.800 | 0.5550 |
| 8 | | 0.02000 | -0.2750 | 0.2750 | 0.5200 | 0.775 | 0.5225 |
| 9 | | 0.02250 | -0.3000 | 0.3000 | 0.4800 | 0.750 | 0.4900 |
| 10 | | 0.02500 | -0.3250 | 0.3250 | 0.4400 | 0.725 | 0.4575 |
| 1 | Input Layer (all connections) | 0.0075 | -0.0750 | 0.0750 | 0.7000 | 0.830 | 0.650 |
| 2 | | 0.0150 | -0.1500 | 0.1500 | 0.6600 | 0.810 | 0.625 |
| 3 | | 0.0225 | -0.2250 | 0.2250 | 0.6200 | 0.790 | 0.600 |
| 4 | | 0.0300 | -0.3000 | 0.3000 | 0.5800 | 0.770 | 0.575 |
| 5 | | 0.0375 | -0.3750 | 0.3750 | 0.5400 | 0.750 | 0.550 |
| 6 | | 0.0450 | -0.4500 | 0.4500 | 0.5000 | 0.730 | 0.525 |
| 7 | | 0.0525 | -0.5250 | 0.5250 | 0.4600 | 0.710 | 0.500 |
| 8 | | 0.0600 | -0.6000 | 0.6000 | 0.4200 | 0.690 | 0.475 |
| 9 | | 0.0675 | -0.6750 | 0.6750 | 0.3800 | 0.670 | 0.450 |
| 10 | | 0.0750 | -0.7500 | 0.7500 | 0.3400 | 0.650 | 0.425 |
| 1 | Hidden Layer Connections | 0.00750 | -0.1000 | 0.1000 | 0.7000 | 0.8250 | 0.5250 |
| 2 | | 0.01500 | -0.1300 | 0.1300 | 0.6500 | 0.8000 | 0.4950 |
| 3 | | 0.02250 | -0.1600 | 0.1600 | 0.6000 | 0.7750 | 0.4650 |
| 4 | | 0.03000 | -0.1900 | 0.1900 | 0.5500 | 0.7500 | 0.4350 |
| 5 | | 0.03750 | -0.2200 | 0.2200 | 0.5000 | 0.7250 | 0.4050 |
| 6 | | 0.04500 | -0.2500 | 0.2500 | 0.4500 | 0.7000 | 0.3750 |
| 7 | | 0.05250 | -0.2800 | 0.2800 | 0.4000 | 0.6750 | 0.3450 |
| 8 | | 0.06000 | -0.3100 | 0.3100 | 0.3500 | 0.6500 | 0.3150 |
| 9 | | 0.06750 | -0.3400 | 0.3400 | 0.3000 | 0.6250 | 0.2850 |
| 10 | | 0.07500 | -0.3700 | 0.3700 | 0.2500 | 0.6000 | 0.2550 |

## D.6 Network B – Glasspool Semantic Design

| | | Lesion Type | | | | | |
|---|---|---|---|---|---|---|---|
| Lesion Severity | Lesion Location | Ablate % | Noise | | Constrain | | Scale |
| | | | Lower | Upper | Lower | Upper | |
| 1 | Randomly through the network | 0.00150 | -0.0700 | 0.0700 | 0.830 | 0.930 | 0.6500 |
| 2 | | 0.00300 | -0.0900 | 0.0900 | 0.790 | 0.910 | 0.6250 |
| 3 | | 0.00450 | -0.1100 | 0.1100 | 0.750 | 0.890 | 0.6000 |
| 4 | | 0.00600 | -0.1300 | 0.1300 | 0.710 | 0.870 | 0.5750 |
| 5 | | 0.00750 | -0.1500 | 0.1500 | 0.670 | 0.850 | 0.5500 |
| 6 | | 0.00900 | -0.1700 | 0.1700 | 0.630 | 0.830 | 0.5250 |
| 7 | | 0.01050 | -0.1900 | 0.1900 | 0.590 | 0.810 | 0.5000 |
| 8 | | 0.01200 | -0.2100 | 0.2100 | 0.550 | 0.790 | 0.4750 |
| 9 | | 0.01350 | -0.2300 | 0.2300 | 0.510 | 0.770 | 0.4500 |
| 10 | | 0.01500 | -0.2500 | 0.2500 | 0.470 | 0.750 | 0.4250 |
| 1 | Input Layer (all connections) | 0.00500 | -0.0600 | 0.0600 | 0.710 | 0.870 | 0.6000 |
| 2 | | 0.01000 | -0.1200 | 0.1200 | 0.670 | 0.850 | 0.5700 |
| 3 | | 0.01500 | -0.1800 | 0.1800 | 0.630 | 0.830 | 0.5400 |
| 4 | | 0.02000 | -0.2400 | 0.2400 | 0.590 | 0.810 | 0.5100 |
| 5 | | 0.02500 | -0.3000 | 0.3000 | 0.550 | 0.790 | 0.4800 |
| 6 | | 0.03000 | -0.3600 | 0.3600 | 0.510 | 0.770 | 0.4500 |
| 7 | | 0.03500 | -0.4200 | 0.4200 | 0.470 | 0.750 | 0.4200 |
| 8 | | 0.04000 | -0.4800 | 0.4800 | 0.430 | 0.730 | 0.3900 |
| 9 | | 0.04500 | -0.5400 | 0.5400 | 0.390 | 0.710 | 0.3600 |
| 10 | | 0.05000 | -0.6000 | 0.6000 | 0.350 | 0.690 | 0.3300 |
| 1 | Hidden Layer Connections | 0.00500 | -0.1000 | 0.1000 | 0.7000 | 0.8250 | 0.5300 |
| 2 | | 0.01000 | -0.1200 | 0.1200 | 0.6500 | 0.8000 | 0.5000 |
| 3 | | 0.01500 | -0.1400 | 0.1400 | 0.6000 | 0.7750 | 0.4700 |
| 4 | | 0.02000 | -0.1600 | 0.1600 | 0.5500 | 0.7500 | 0.4400 |
| 5 | | 0.02500 | -0.1800 | 0.1800 | 0.5000 | 0.7250 | 0.4100 |
| 6 | | 0.03000 | -0.2000 | 0.2000 | 0.4500 | 0.7000 | 0.3800 |
| 7 | | 0.03500 | -0.2200 | 0.2200 | 0.4000 | 0.6750 | 0.3500 |
| 8 | | 0.04000 | -0.2400 | 0.2400 | 0.3500 | 0.6500 | 0.3200 |
| 9 | | 0.04500 | -0.2600 | 0.2600 | 0.3000 | 0.6250 | 0.2900 |
| 10 | | 0.05000 | -0.2800 | 0.2800 | 0.2500 | 0.6000 | 0.2600 |

# Appendix E Sample Error Data.

The following table contains a sample of raw data used to calculate the magnitude, and used to determine statistical difference. Similar raw data would exist for errors associated with low and high, and complex and simple CV words.

This raw data comes from comparing abstract and concrete errors resulting from ablating hidden layer connections on network B using the Glasspool semantic vector design.

| Train Seed | Recall Seed | Severity | Abstract Errors | Concrete Errors | Abstract Words | Concrete Words | Abstract % | Concrete % |
|---|---|---|---|---|---|---|---|---|
| 123 | 10001 | 1 | 14 | 6 | 200 | 200 | 7.00% | 3.00% |
| 123 | 10001 | 2 | 37 | 20 | 200 | 200 | 18.50% | 10.00% |
| 123 | 10001 | 3 | 55 | 59 | 200 | 200 | 27.50% | 29.50% |
| 123 | 10001 | 4 | 69 | 73 | 200 | 200 | 34.50% | 36.50% |
| 123 | 10001 | 5 | 97 | 94 | 200 | 200 | 48.50% | 47.00% |
| 123 | 10001 | 6 | 113 | 125 | 200 | 200 | 56.50% | 62.50% |
| 123 | 10001 | 7 | 129 | 133 | 200 | 200 | 64.50% | 66.50% |
| 123 | 10001 | 8 | 143 | 145 | 200 | 200 | 71.50% | 72.50% |
| 123 | 10001 | 9 | 156 | 160 | 200 | 200 | 78.00% | 80.00% |
| 123 | 10001 | 10 | 164 | 171 | 200 | 200 | 82.00% | 85.50% |
| 123 | 10002 | 1 | 11 | 9 | 200 | 200 | 5.50% | 4.50% |
| 123 | 10002 | 2 | 24 | 19 | 200 | 200 | 12.00% | 9.50% |
| 123 | 10002 | 3 | 43 | 44 | 200 | 200 | 21.50% | 22.00% |
| 123 | 10002 | 4 | 98 | 61 | 200 | 200 | 49.00% | 30.50% |
| 123 | 10002 | 5 | 120 | 85 | 200 | 200 | 60.00% | 42.50% |
| 123 | 10002 | 6 | 132 | 103 | 200 | 200 | 66.00% | 51.50% |
| 123 | 10002 | 7 | 145 | 110 | 200 | 200 | 72.50% | 55.00% |
| 123 | 10002 | 8 | 152 | 124 | 200 | 200 | 76.00% | 62.00% |
| 123 | 10002 | 9 | 171 | 131 | 200 | 200 | 85.50% | 65.50% |
| 123 | 10002 | 10 | 174 | 152 | 200 | 200 | 87.00% | 76.00% |
| 123 | 10003 | 1 | 19 | 17 | 200 | 200 | 9.50% | 8.50% |
| 123 | 10003 | 2 | 28 | 30 | 200 | 200 | 14.00% | 15.00% |
| 123 | 10003 | 3 | 63 | 48 | 200 | 200 | 31.50% | 24.00% |
| 123 | 10003 | 4 | 75 | 71 | 200 | 200 | 37.50% | 35.50% |
| 123 | 10003 | 5 | 125 | 87 | 200 | 200 | 62.50% | 43.50% |
| 123 | 10003 | 6 | 148 | 111 | 200 | 200 | 74.00% | 55.50% |
| 123 | 10003 | 7 | 151 | 118 | 200 | 200 | 75.50% | 59.00% |
| 123 | 10003 | 8 | 161 | 125 | 200 | 200 | 80.50% | 62.50% |
| 123 | 10003 | 9 | 165 | 135 | 200 | 200 | 82.50% | 67.50% |
| 123 | 10003 | 10 | 169 | 137 | 200 | 200 | 84.50% | 68.50% |
| 123 | 10004 | 1 | 11 | 15 | 200 | 200 | 5.50% | 7.50% |
| 123 | 10004 | 2 | 27 | 18 | 200 | 200 | 13.50% | 9.00% |
| 123 | 10004 | 3 | 72 | 38 | 200 | 200 | 36.00% | 19.00% |
| 123 | 10004 | 4 | 79 | 51 | 200 | 200 | 39.50% | 25.50% |
| 123 | 10004 | 5 | 94 | 71 | 200 | 200 | 47.00% | 35.50% |
| 123 | 10004 | 6 | 108 | 93 | 200 | 200 | 54.00% | 46.50% |
| 123 | 10004 | 7 | 129 | 105 | 200 | 200 | 64.50% | 52.50% |
| 123 | 10004 | 8 | 136 | 124 | 200 | 200 | 68.00% | 62.00% |
| 123 | 10004 | 9 | 159 | 131 | 200 | 200 | 79.50% | 65.50% |
| 123 | 10004 | 10 | 178 | 144 | 200 | 200 | 89.00% | 72.00% |
| 123 | 10005 | 1 | 6 | 8 | 200 | 200 | 3.00% | 4.00% |
| 123 | 10005 | 2 | 27 | 18 | 200 | 200 | 13.50% | 9.00% |
| 123 | 10005 | 3 | 45 | 55 | 200 | 200 | 22.50% | 27.50% |
| 123 | 10005 | 4 | 81 | 46 | 200 | 200 | 40.50% | 23.00% |

| 123 | 10005 | 5 | 106 | 70 | 200 | 200 | 53.00% | 35.00% |
|---|---|---|---|---|---|---|---|---|
| 123 | 10005 | 6 | 130 | 141 | 200 | 200 | 65.00% | 70.50% |
| 123 | 10005 | 7 | 129 | 143 | 200 | 200 | 64.50% | 71.50% |
| 123 | 10005 | 8 | 144 | 132 | 200 | 200 | 72.00% | 66.00% |
| 123 | 10005 | 9 | 152 | 159 | 200 | 200 | 76.00% | 79.50% |
| 123 | 10005 | 10 | 166 | 155 | 200 | 200 | 83.00% | 77.50% |
| 234 | 10001 | 1 | 17 | 10 | 200 | 200 | 8.50% | 5.00% |
| 234 | 10001 | 2 | 27 | 29 | 200 | 200 | 13.50% | 14.50% |
| 234 | 10001 | 3 | 67 | 41 | 200 | 200 | 33.50% | 20.50% |
| 234 | 10001 | 4 | 92 | 50 | 200 | 200 | 46.00% | 25.00% |
| 234 | 10001 | 5 | 107 | 77 | 200 | 200 | 53.50% | 38.50% |
| 234 | 10001 | 6 | 143 | 87 | 200 | 200 | 71.50% | 43.50% |
| 234 | 10001 | 7 | 145 | 105 | 200 | 200 | 72.50% | 52.50% |
| 234 | 10001 | 8 | 160 | 123 | 200 | 200 | 80.00% | 61.50% |
| 234 | 10001 | 9 | 172 | 130 | 200 | 200 | 86.00% | 65.00% |
| 234 | 10001 | 10 | 170 | 136 | 200 | 200 | 85.00% | 68.00% |
| 234 | 10002 | 1 | 23 | 7 | 200 | 200 | 11.50% | 3.50% |
| 234 | 10002 | 2 | 53 | 26 | 200 | 200 | 26.50% | 13.00% |
| 234 | 10002 | 3 | 60 | 37 | 200 | 200 | 30.00% | 18.50% |
| 234 | 10002 | 4 | 98 | 52 | 200 | 200 | 49.00% | 26.00% |
| 234 | 10002 | 5 | 114 | 71 | 200 | 200 | 57.00% | 35.50% |
| 234 | 10002 | 6 | 124 | 81 | 200 | 200 | 62.00% | 40.50% |
| 234 | 10002 | 7 | 137 | 98 | 200 | 200 | 68.50% | 49.00% |
| 234 | 10002 | 8 | 150 | 112 | 200 | 200 | 75.00% | 56.00% |
| 234 | 10002 | 9 | 158 | 123 | 200 | 200 | 79.00% | 61.50% |
| 234 | 10002 | 10 | 178 | 143 | 200 | 200 | 89.00% | 71.50% |
| 234 | 10003 | 1 | 12 | 9 | 200 | 200 | 6.00% | 4.50% |
| 234 | 10003 | 2 | 19 | 17 | 200 | 200 | 9.50% | 8.50% |
| 234 | 10003 | 3 | 61 | 37 | 200 | 200 | 30.50% | 18.50% |
| 234 | 10003 | 4 | 89 | 60 | 200 | 200 | 44.50% | 30.00% |
| 234 | 10003 | 5 | 91 | 82 | 200 | 200 | 45.50% | 41.00% |
| 234 | 10003 | 6 | 93 | 97 | 200 | 200 | 46.50% | 48.50% |
| 234 | 10003 | 7 | 124 | 104 | 200 | 200 | 62.00% | 52.00% |
| 234 | 10003 | 8 | 132 | 109 | 200 | 200 | 66.00% | 54.50% |
| 234 | 10003 | 9 | 160 | 126 | 200 | 200 | 80.00% | 63.00% |
| 234 | 10003 | 10 | 170 | 126 | 200 | 200 | 85.00% | 63.00% |
| 234 | 10004 | 1 | 11 | 10 | 200 | 200 | 5.50% | 5.00% |
| 234 | 10004 | 2 | 37 | 14 | 200 | 200 | 18.50% | 7.00% |
| 234 | 10004 | 3 | 78 | 32 | 200 | 200 | 39.00% | 16.00% |
| 234 | 10004 | 4 | 76 | 60 | 200 | 200 | 38.00% | 30.00% |
| 234 | 10004 | 5 | 89 | 70 | 200 | 200 | 44.50% | 35.00% |
| 234 | 10004 | 6 | 122 | 80 | 200 | 200 | 61.00% | 40.00% |
| 234 | 10004 | 7 | 140 | 110 | 200 | 200 | 70.00% | 55.00% |
| 234 | 10004 | 8 | 140 | 114 | 200 | 200 | 70.00% | 57.00% |
| 234 | 10004 | 9 | 161 | 129 | 200 | 200 | 80.50% | 64.50% |
| 234 | 10004 | 10 | 160 | 137 | 200 | 200 | 80.00% | 68.50% |
| 234 | 10005 | 1 | 12 | 4 | 200 | 200 | 6.00% | 2.00% |
| 234 | 10005 | 2 | 31 | 16 | 200 | 200 | 15.50% | 8.00% |
| 234 | 10005 | 3 | 58 | 29 | 200 | 200 | 29.00% | 14.50% |
| 234 | 10005 | 4 | 89 | 42 | 200 | 200 | 44.50% | 21.00% |
| 234 | 10005 | 5 | 103 | 60 | 200 | 200 | 51.50% | 30.00% |
| 234 | 10005 | 6 | 129 | 92 | 200 | 200 | 64.50% | 46.00% |
| 234 | 10005 | 7 | 145 | 108 | 200 | 200 | 72.50% | 54.00% |
| 234 | 10005 | 8 | 140 | 111 | 200 | 200 | 70.00% | 55.50% |
| 234 | 10005 | 9 | 164 | 125 | 200 | 200 | 82.00% | 62.50% |
| 234 | 10005 | 10 | 166 | 121 | 200 | 200 | 83.00% | 60.50% |

| 345 | 10001 | 1 | 2 | 8 | 200 | 200 | 1.00% | 4.00% |
|---|---|---|---|---|---|---|---|---|
| 345 | 10001 | 2 | 28 | 40 | 200 | 200 | 14.00% | 20.00% |
| 345 | 10001 | 3 | 35 | 50 | 200 | 200 | 17.50% | 25.00% |
| 345 | 10001 | 4 | 52 | 55 | 200 | 200 | 26.00% | 27.50% |
| 345 | 10001 | 5 | 81 | 61 | 200 | 200 | 40.50% | 30.50% |
| 345 | 10001 | 6 | 86 | 69 | 200 | 200 | 43.00% | 34.50% |
| 345 | 10001 | 7 | 107 | 90 | 200 | 200 | 53.50% | 45.00% |
| 345 | 10001 | 8 | 122 | 93 | 200 | 200 | 61.00% | 46.50% |
| 345 | 10001 | 9 | 153 | 116 | 200 | 200 | 76.50% | 58.00% |
| 345 | 10001 | 10 | 158 | 138 | 200 | 200 | 79.00% | 69.00% |
| 345 | 10002 | 1 | 17 | 10 | 200 | 200 | 8.50% | 5.00% |
| 345 | 10002 | 2 | 30 | 23 | 200 | 200 | 15.00% | 11.50% |
| 345 | 10002 | 3 | 32 | 33 | 200 | 200 | 16.00% | 16.50% |
| 345 | 10002 | 4 | 79 | 55 | 200 | 200 | 39.50% | 27.50% |
| 345 | 10002 | 5 | 122 | 71 | 200 | 200 | 61.00% | 35.50% |
| 345 | 10002 | 6 | 129 | 96 | 200 | 200 | 64.50% | 48.00% |
| 345 | 10002 | 7 | 153 | 94 | 200 | 200 | 76.50% | 47.00% |
| 345 | 10002 | 8 | 165 | 100 | 200 | 200 | 82.50% | 50.00% |
| 345 | 10002 | 9 | 169 | 120 | 200 | 200 | 84.50% | 60.00% |
| 345 | 10002 | 10 | 168 | 129 | 200 | 200 | 84.00% | 64.50% |
| 345 | 10003 | 1 | 10 | 16 | 200 | 200 | 5.00% | 8.00% |
| 345 | 10003 | 2 | 30 | 35 | 200 | 200 | 15.00% | 17.50% |
| 345 | 10003 | 3 | 44 | 33 | 200 | 200 | 22.00% | 16.50% |
| 345 | 10003 | 4 | 60 | 57 | 200 | 200 | 30.00% | 28.50% |
| 345 | 10003 | 5 | 91 | 69 | 200 | 200 | 45.50% | 34.50% |
| 345 | 10003 | 6 | 111 | 82 | 200 | 200 | 55.50% | 41.00% |
| 345 | 10003 | 7 | 126 | 90 | 200 | 200 | 63.00% | 45.00% |
| 345 | 10003 | 8 | 138 | 91 | 200 | 200 | 69.00% | 45.50% |
| 345 | 10003 | 9 | 152 | 115 | 200 | 200 | 76.00% | 57.50% |
| 345 | 10003 | 10 | 148 | 135 | 200 | 200 | 74.00% | 67.50% |
| 345 | 10004 | 1 | 6 | 4 | 200 | 200 | 3.00% | 2.00% |
| 345 | 10004 | 2 | 30 | 20 | 200 | 200 | 15.00% | 10.00% |
| 345 | 10004 | 3 | 58 | 31 | 200 | 200 | 29.00% | 15.50% |
| 345 | 10004 | 4 | 90 | 44 | 200 | 200 | 45.00% | 22.00% |
| 345 | 10004 | 5 | 108 | 57 | 200 | 200 | 54.00% | 28.50% |
| 345 | 10004 | 6 | 122 | 58 | 200 | 200 | 61.00% | 29.00% |
| 345 | 10004 | 7 | 146 | 72 | 200 | 200 | 73.00% | 36.00% |
| 345 | 10004 | 8 | 157 | 102 | 200 | 200 | 78.50% | 51.00% |
| 345 | 10004 | 9 | 161 | 112 | 200 | 200 | 80.50% | 56.00% |
| 345 | 10004 | 10 | 163 | 125 | 200 | 200 | 81.50% | 62.50% |
| 345 | 10005 | 1 | 10 | 10 | 200 | 200 | 5.00% | 5.00% |
| 345 | 10005 | 2 | 33 | 20 | 200 | 200 | 16.50% | 10.00% |
| 345 | 10005 | 3 | 66 | 47 | 200 | 200 | 33.00% | 23.50% |
| 345 | 10005 | 4 | 80 | 45 | 200 | 200 | 40.00% | 22.50% |
| 345 | 10005 | 5 | 102 | 65 | 200 | 200 | 51.00% | 32.50% |
| 345 | 10005 | 6 | 125 | 99 | 200 | 200 | 62.50% | 49.50% |
| 345 | 10005 | 7 | 134 | 113 | 200 | 200 | 67.00% | 56.50% |
| 345 | 10005 | 8 | 134 | 119 | 200 | 200 | 67.00% | 59.50% |
| 345 | 10005 | 9 | 163 | 137 | 200 | 200 | 81.50% | 68.50% |
| 345 | 10005 | 10 | 170 | 141 | 200 | 200 | 85.00% | 70.50% |
| Totals | | | | | | | 7591.50% | 5892.00% |

$$m = \frac{\sum(\beta - \alpha)}{N} = \frac{5892.00 - 7591.50}{150} = -11.33\%$$

# Appendix F Plaut & Shallice (1993) Corpus

| Word | Attribute Number |
|---|---|
| TART | 100010111000010010110011010000000000000000000000000010100101001000000001000000000000000000000000000 |
| TENT | 001001001000000100100001000110110000000000000000000011010010001000100001000000000001001010000000000 |
| FACE | 100010100000001000101100011100000000111110000100000000000000000000100000000001000000000000000010000 |
| DEER | 001001010101010010101001000110110000001101001100000000100000000001000000100000000000000000000000000 |
| COAT | 010010100011000110100000011100000000110000000000000011100100100000000000000000000000000000000000000 |
| GRIN | 100010001000001000101100011100000000110000000110000000000000000000100010000000000001000000000000000 |
| LOCK | 100001100000000100100010001100000011100000000000010000000100010010000000000000000000000000010000000000 |
| ROPE | 001100010000110001000000001100000101100000000000000011000010001001100000000000000000000000001000000000 |
| HARE | 010001010100010010101001000100011000001101001100000000100001000001000000000000000000000000000000000000 |
| LASS | 010001100100001000101101001100000000001100011100000001000000001001100000000000000000000000000000000000 |
| FLAN | 100100011000000010010011010000000000000000000000000010100101001000000001000000000000000000000000000000 |
| HIND | 010001010101010010101001000110110000001101001100000000000000000000101000010000000000000000000000000000 |
| WAVE | 001001001000000101010100001010100000000010010000000100000000000000010000001000000000010000000000000000 |
| FLEA | 100001001100000000101110110100000100010100110011000000011000000100000001000000000000000000000000000000 |
| STAR | 010010100001000001010000011000000000000010010000001000100000000001000000000100000000000000000000000000 |
| REED | 001100100000001000101001000101100000001001000101000000000000100000000000000000000000000000000000000000 |
| LOON | 010001010001001010101000010100110000001011110110000001000000000000000000000000000000000000000000000000 |
| CASE | 010001100000000101000101001000000110000000000000001000001100101000000000000000000000000000100000000 |
| FLAG | 010010100000000100101000011000000000000000000000000011010010001000100000000000100000000000000000000 |
| POST | 010100110000000101000001001000001001000000000000101100001000100010000000000000000000000000000000000000 |
| TACT | 000000000000000000000000000000000000000000000000000000000000000000000000000000000100100000000000000000001011010 |
| RENT | 000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000001100101000 |
| FACT | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000101000000010000000000000 |
| DEED | 000000000000000000000000000000000000000000000000000000000000000000000000000000001010100000000011100000000001001 |
| COST | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000000000000000010001000000 |
| GAIN | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000000000000101001000000000 |
| LACK | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000000100000001000000000000 |
| ROLE | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000010000000000010000000000 |
| HIRE | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000000010001000111110100000 |
| LOSS | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000000000000101001000000000 |
| PLAN | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000001100001001000000000100 |
| HINT | 000000000000000000000000000000000000000000000000000000000000000000000000000000010000011000001001000000010000 |

| | |
|---|---|
| WAGE | 0000000000

# Appendix G Sample Configuration File

The following shows a number of modifiable items provided in the configuration file. Any line prefixed by a '#' character is ignored and for documentation purposes only. Note that some of these values can be assigned through the GBDS command line interface. However, if these are set at the environment level (e.g. using the 'set' command in DOS or UNIX), then they will override any command line values.

```
# Maximum Word Length
MAX_WORDLEN        15

# Ignore doubles if they are specified in the corpus file
#
# 1 = TRUE
# 0 = FALSE
#
IGNORE_DOUBLES     1

#
# Save analysis summary to file
#
ANALYSIS_SUMMARY         1
ANALYSIS_SUMMARY_FILE    AnalysisSummary

#
# File holding behaviour of learning success
#
LEARN_SUCCESS_FILE       LearnSuccess.txt

#
# Save Learning trace to file
#
LEARNING_TRACE           1
LEARNING_TRACE_FILE      LearningTrace

#
# Letter Threshold training margin
#
# This value is added to the Letter Threshold during training.
# This 'over-training' prevents spurious errors due to model learning
# to exact values, and then throwing many errors during recall
#
LETTER_THRESHOLD_TRAINING_MARGIN     0.025

#
# Omit Threshold training margin
#
# This value is substracted from the Omit Threshold during training.
#
OMIT_THRESHOLD_TRAINING_MARGIN       0.025

#
# Stop Threshold training margin
#
# This value is substracted from the Stop Threshold during training.
#
STOP_THRESHOLD_TRAINING_MARGIN       0.025
```

```
#
# Use random threshold margins
#
# Possibly more plausible than a fixed threshold margin
#
# 1 = TRUE
# 0 = FALSE
#
# If this is true, then the LETTER and STOP margins are a random value
# between 0 and the specified value
#USE_RANDOM_THRESHOLD_MARGINS       1
USE_RANDOM_THRESHOLD_MARGINS        0


################################################
# Use different Learning and Recall Thresholds#
################################################

#
# Learn Thresholds
#

# Default threshold for letters and geminates
DEFAULT_LEARN_LETTER_THRESHOLD      0.8
# Default threshold for geminate values
DEFAULT_LEARN_GEMINATE_THRESHOLD    0.8
# Default threshold for unknown letter
# DEFAULT_LEARN_UNKNOWN_THRESHOLD   0.6
# Default threshold for stop value
DEFAULT_LEARN_STOP_THRESHOLD  0.60

#
# Recall Thresholds
#

# Default threshold for letters and geminates
DEFAULT_RECALL_LETTER_THRESHOLD     0.8
# Default threshold for geminate values
DEFAULT_RECALL_GEMINATE_THRESHOLD   0.8
# Default threshold for unknown letter
# DEFAULT_RECALL_UNKNOWN_THRESHOLD  0.6
# Default threshold for omit letter
# DEFAULT_RECALL_OMIT_THRESHOLD  0.60
# Default threshold for stop value
DEFAULT_RECALL_STOP_THRESHOLD  0.60

# List of letter thresholds to use on an individual basis
# caters for the possibility that certain letters may be more
# difficult to learn than others
#
# It is unnecessary to specify all of these.  If you omit one
# that letter will take the value of DEFAULT_LETTER_THRESHOLD
#
# Format is 'LEARN_LETTER_THRESHOLD_x' or RECALL_LETTER_THRESHOLD_x
where 'x' is the letter A-Z
#LEARN_LETTER_THRESHOLD_A     0.8
#LEARN_LETTER_THRESHOLD_B     0.8
#LEARN_LETTER_THRESHOLD_C     0.8
#LEARN_LETTER_THRESHOLD_D     0.8
#LEARN_LETTER_THRESHOLD_E     0.8
```

```
#LEARN_LETTER_THRESHOLD_F      0.8
#LEARN_LETTER_THRESHOLD_G      0.8
#LEARN_LETTER_THRESHOLD_H      0.8
#LEARN_LETTER_THRESHOLD_I      0.8
#LEARN_LETTER_THRESHOLD_J      0.8
#LEARN_LETTER_THRESHOLD_K      0.8
#LEARN_LETTER_THRESHOLD_L      0.8
#LEARN_LETTER_THRESHOLD_M      0.8
#LEARN_LETTER_THRESHOLD_N      0.8
#LEARN_LETTER_THRESHOLD_O      0.8
#LEARN_LETTER_THRESHOLD_P      0.8
#LEARN_LETTER_THRESHOLD_Q      0.8
#LEARN_LETTER_THRESHOLD_R      0.8
#LEARN_LETTER_THRESHOLD_S      0.8
#LEARN_LETTER_THRESHOLD_T      0.8
#LEARN_LETTER_THRESHOLD_U      0.8
#LEARN_LETTER_THRESHOLD_V      0.8
#LEARN_LETTER_THRESHOLD_W      0.8
#LEARN_LETTER_THRESHOLD_X      0.8
#LEARN_LETTER_THRESHOLD_Y      0.8
#LEARN_LETTER_THRESHOLD_Z      0.8

# List of geminate thresholds to use on an individual basis
# caters for the possibility that certain letters may be easier
# to double than others
#
# It is unnecessary to specify all of these.  If you omit one
# that letter will take the value of DEFAULT_GEMINATE_THRESHOLD
#
# Format is 'LEARN_GEMINATE_THRESHOLD_x' or
# RECALL_GEMINATE_THRESHOLD_x where 'x' is the letter A-Z
#LEARN_GEMINATE_THRESHOLD_A    0.8
#LEARN_GEMINATE_THRESHOLD_B    0.8
#LEARN_GEMINATE_THRESHOLD_C    0.8
#LEARN_GEMINATE_THRESHOLD_D    0.8
#LEARN_GEMINATE_THRESHOLD_E    0.8
#LEARN_GEMINATE_THRESHOLD_F    0.8
#LEARN_GEMINATE_THRESHOLD_G    0.8
#LEARN_GEMINATE_THRESHOLD_H    0.8
#LEARN_GEMINATE_THRESHOLD_I    0.8
#LEARN_GEMINATE_THRESHOLD_J    0.8
#LEARN_GEMINATE_THRESHOLD_K    0.8
#LEARN_GEMINATE_THRESHOLD_L    0.8
#LEARN_GEMINATE_THRESHOLD_M    0.8
#LEARN_GEMINATE_THRESHOLD_N    0.8
#LEARN_GEMINATE_THRESHOLD_O    0.8
#LEARN_GEMINATE_THRESHOLD_P    0.8
#LEARN_GEMINATE_THRESHOLD_Q    0.8
#LEARN_GEMINATE_THRESHOLD_R    0.8
#LEARN_GEMINATE_THRESHOLD_S    0.8
#LEARN_GEMINATE_THRESHOLD_T    0.8
#LEARN_GEMINATE_THRESHOLD_U    0.8
#LEARN_GEMINATE_THRESHOLD_V    0.8
#LEARN_GEMINATE_THRESHOLD_W    0.8
#LEARN_GEMINATE_THRESHOLD_X    0.8
#LEARN_GEMINATE_THRESHOLD_Y    0.8
#LEARN_GEMINATE_THRESHOLD_Z    0.8

# Set an 'unknown' threshold per letter.  This is the level below
# which we know that a letter is there, but we're not quite sure what
# it is
```

```
#
# It is unnecessary to specify all of these.  If you omit one
# that letter will take the value of DEFAULT_UNKNOWN_THRESHOLD
#
# Format is 'LEARN_UNKNOWN_THRESHOLD_x' or RECALL_UNKNOWN_THRESHOLD_x
where 'x' is the letter A-Z
#LEARN_UNKNOWN_THRESHOLD_A     0.6
#LEARN_UNKNOWN_THRESHOLD_B     0.6
#LEARN_UNKNOWN_THRESHOLD_C     0.6
#LEARN_UNKNOWN_THRESHOLD_D     0.6
#LEARN_UNKNOWN_THRESHOLD_E     0.6
#LEARN_UNKNOWN_THRESHOLD_F     0.6
#LEARN_UNKNOWN_THRESHOLD_G     0.6
#LEARN_UNKNOWN_THRESHOLD_H     0.6
#LEARN_UNKNOWN_THRESHOLD_I     0.6
#LEARN_UNKNOWN_THRESHOLD_J     0.6
#LEARN_UNKNOWN_THRESHOLD_K     0.6
#LEARN_UNKNOWN_THRESHOLD_L     0.6
#LEARN_UNKNOWN_THRESHOLD_M     0.6
#LEARN_UNKNOWN_THRESHOLD_N     0.6
#LEARN_UNKNOWN_THRESHOLD_O     0.6
#LEARN_UNKNOWN_THRESHOLD_P     0.6
#LEARN_UNKNOWN_THRESHOLD_Q     0.6
#LEARN_UNKNOWN_THRESHOLD_R     0.6
#LEARN_UNKNOWN_THRESHOLD_S     0.6
#LEARN_UNKNOWN_THRESHOLD_T     0.6
#LEARN_UNKNOWN_THRESHOLD_U     0.6
#LEARN_UNKNOWN_THRESHOLD_V     0.6
#LEARN_UNKNOWN_THRESHOLD_W     0.6
#LEARN_UNKNOWN_THRESHOLD_X     0.6
#LEARN_UNKNOWN_THRESHOLD_Y     0.6
#LEARN_UNKNOWN_THRESHOLD_Z     0.6

# Set an omit threshold per letter.  This is the level below which
# we should ignore the recall of a letter
#
# It is unnecessary to specify all of these.  If you omit one
# that letter will take the value of DEFAULT_OMIT_THRESHOLD
#
# Format is 'LEARN_OMIT_THRESHOLD_x' or RECALL_OMIT_THRESHOLD_x where
# 'x' is the letter A-Z
#LEARN_OMIT_THRESHOLD_A 0.6
#LEARN_OMIT_THRESHOLD_B 0.6
#LEARN_OMIT_THRESHOLD_C 0.6
#LEARN_OMIT_THRESHOLD_D 0.6
#LEARN_OMIT_THRESHOLD_E 0.6
#LEARN_OMIT_THRESHOLD_F 0.6
#LEARN_OMIT_THRESHOLD_G 0.6
#LEARN_OMIT_THRESHOLD_H 0.6
#LEARN_OMIT_THRESHOLD_I 0.6
#LEARN_OMIT_THRESHOLD_J 0.6
#LEARN_OMIT_THRESHOLD_K 0.6
#LEARN_OMIT_THRESHOLD_L 0.6
#LEARN_OMIT_THRESHOLD_M 0.6
#LEARN_OMIT_THRESHOLD_N 0.6
#LEARN_OMIT_THRESHOLD_O 0.6
#LEARN_OMIT_THRESHOLD_P 0.6
#LEARN_OMIT_THRESHOLD_Q 0.6
#LEARN_OMIT_THRESHOLD_R 0.6
#LEARN_OMIT_THRESHOLD_S 0.6
#LEARN_OMIT_THRESHOLD_T 0.6
```

```
#LEARN_OMIT_THRESHOLD_U 0.6
#LEARN_OMIT_THRESHOLD_V 0.6
#LEARN_OMIT_THRESHOLD_W 0.6
#LEARN_OMIT_THRESHOLD_X 0.6
#LEARN_OMIT_THRESHOLD_Y 0.6
#LEARN_OMIT_THRESHOLD_Z 0.6

# Set a stop threshold per letter.  This is the level below which
# we consider recall terminated
#
# It is unnecessary to specify all of these.  If you omit one
# that letter will take the value of DEFAULT_STOP_THRESHOLD
#
# Format is 'LEARN_STOP_THRESHOLD_x' or RECALL_STOP_THRESHOLD_x where
# 'x' is the letter A-Z
#LEARN_STOP_THRESHOLD_A 0.6
#LEARN_STOP_THRESHOLD_B 0.6
#LEARN_STOP_THRESHOLD_C 0.6
#LEARN_STOP_THRESHOLD_D 0.6
#LEARN_STOP_THRESHOLD_E 0.6
#LEARN_STOP_THRESHOLD_F 0.6
#LEARN_STOP_THRESHOLD_G 0.6
#LEARN_STOP_THRESHOLD_H 0.6
#LEARN_STOP_THRESHOLD_I 0.6
#LEARN_STOP_THRESHOLD_J 0.6
#LEARN_STOP_THRESHOLD_K 0.6
#LEARN_STOP_THRESHOLD_L 0.6
#LEARN_STOP_THRESHOLD_M 0.6
#LEARN_STOP_THRESHOLD_N 0.6
#LEARN_STOP_THRESHOLD_O 0.6
#LEARN_STOP_THRESHOLD_P 0.6
#LEARN_STOP_THRESHOLD_Q 0.6
#LEARN_STOP_THRESHOLD_R 0.6
#LEARN_STOP_THRESHOLD_S 0.6
#LEARN_STOP_THRESHOLD_T 0.6
#LEARN_STOP_THRESHOLD_U 0.6
#LEARN_STOP_THRESHOLD_V 0.6
#LEARN_STOP_THRESHOLD_W 0.6
#LEARN_STOP_THRESHOLD_X 0.6
#LEARN_STOP_THRESHOLD_Y 0.6
#LEARN_STOP_THRESHOLD_Z 0.6

# What is value to use for an 'ON' and 'OFF' states.  Instead of a
standard 1.0/0.0
# it might be interesting to play with other values
ON_VALUE          1.00
OFF_VALUE         0.00

# When considering simple/complex CV
#
# Can non-even length words be considered simple-cv
#     e.g. CVCVC as well as CVCV or CVCVCV
ALLOW_ODD_SIMPLE_CV     0

#
#     Is Y considered a Consonant or a VOWEL
#
#     1 - Yes Y is a vowel
#     0 - No Y is a consonant
Y_IS_A_VOWEL      0
```

```
# How many stop tokens to learn the end of a word?
#
STOPSTATES        2

# Use Consonant Vowel Attributes
USINGCV           1

# Are we testing Geminates
USINGGEMINATE     1

# Default Learning Rate
LEARNING_RATE     0.01

# Recovery FACTOR
# In order to keep the activations between the sigmoid boundaries
# (0.0 -> 1.0),
# scale the output by a function designed to impede recent characters
# by a value proportional to the recency of that letter's appearance
#
# distance = num characters since this letter last seen
# scale = (1.0 - pow(FACTOR, distance))
#
# Actual output is then multiplied by this scale
#
# The impact of a lower FACTOR is to increase the slope (i.e.
# recovery) of the impact on the activation
RECOVERY_FACTOR   0.05

# Default context type
# 1 - Positional context
# 2 - IE Context
# 3 - Sliding Window Context
CONTEXTTYPE       3

# IE Factor used in calculating exponential curves
# Used for IE context
IE_FACTOR         0.5

# Sliding window size
# Used for sliding window context
SWINDOW_SIZE      8

# When learning words, choose them in random order
# 1 = TRUE
# 0 = FALSE
#
# If you choose FALSE, then the words will always be presented to
# the system in the order that they are listed in the input file
RANDOMWORDORDER   1

# If stopping when 'Good enough' i.e. system seems to have learned
# everything 100%, then how many epochs need to show 100% correct
# behaviour?
GOOD_ENOUGH_EPOCHS      20

# Default number of hidden nodes in the Letter Network
LETNET_HIDDEN     25

#
# For the Semantic Backprop network, how often should high/low words
# be presented?
```

```
#
HIGH_FREQ_PRESENT       1.0
LOW_FREQ_PRESENT        0.3

#
#     Letter Network Lesion Locations
#
#     0 - Randomly through the network
#     1 - Input layer to Hidden
#     2 - Hidden Layer to Output
#
LESION_LOCN       0

#
# If lesioning from the input layer to the hidden layer, how should
# it be divided?  It's possible to selectively lesion the context or
# semantic nodes
#
# When lesioning the letter network, should the connections from the
# context bits be lesioned?
LESION_CONTEXT    1

# When lesioning the letter network, should the connections from the
# semantic nodes be lesioned?
LESION_SEMANTICS  1

#
# When counting errors, should the system collate any errors beyond
# the end of the word?  Therefore counting them as if they were in the
# last position?
#
COLLATE_TRAILING  0
```

# Appendix H Examination of Semantic Designs

In Chapter 6, we provided evidence that the Glasspool et al semantic design did not consistently show abstract words as being more erroneous than concrete words. For the sake of brevity, we have deferred a more thorough investigation to this appendix.

Chapter 5 discussed how abstract and concrete attributes for semantic vectors in the GSC model are assigned predefined positions. Of the 56 attribute vectors, 28 are labelled *concrete* and 28 are labelled *abstract*. Concrete words use more active features than abstract words, but are only located in half of the vector. We believe that for a standard feed forward algorithm, using 1, and 0 as ON/OFF values with the Glasspool design (B-DG) is not as robust in the presence of damage as the Plaut & Shallice approach (B-PS). Despite the *number* of errors being similar across both approaches, the B-DG vector design seems to break down more easily in the sense that under certain circumstances, concrete words are more erroneous that abstract words, and the locus of impairment where this is most likely to occur is between the input and hidden layer. In a forward pass, the net input $net_i$ to node $j$ in the hidden or output layer is given by:

$$net_i = \sum_{j=1}^{n} A_j W_{ji} \qquad \textbf{Equation H.1}$$

where $A_j$ is the activation of node $j$ in the previous layer and $W_{ji}$ is the weight from node $j$ to node $i$. When computing $net_j$ in the hidden layer, $A_j$ is the value 0 or 1 provided by the input vector attributes. Clearly lesioning a connection with an input value of 0 will have no effect on weights connected to that node as they are multiplied by 0. We believe it is important to distinguish between an OFF value and no input whatsoever, and that patterns consistently assigning no input to an attribute position are likely to affect the ability of the network to gracefully degrade when damaged.

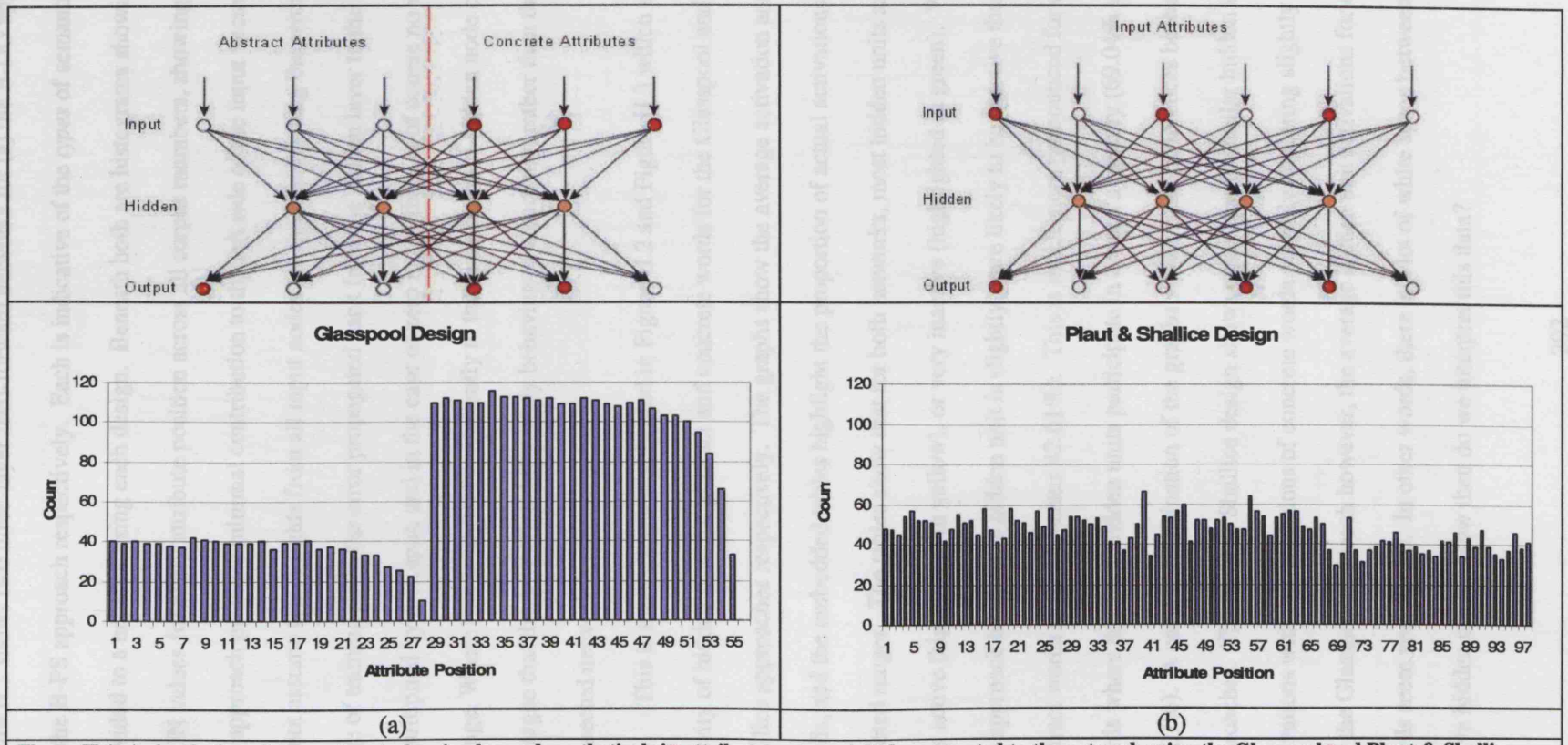
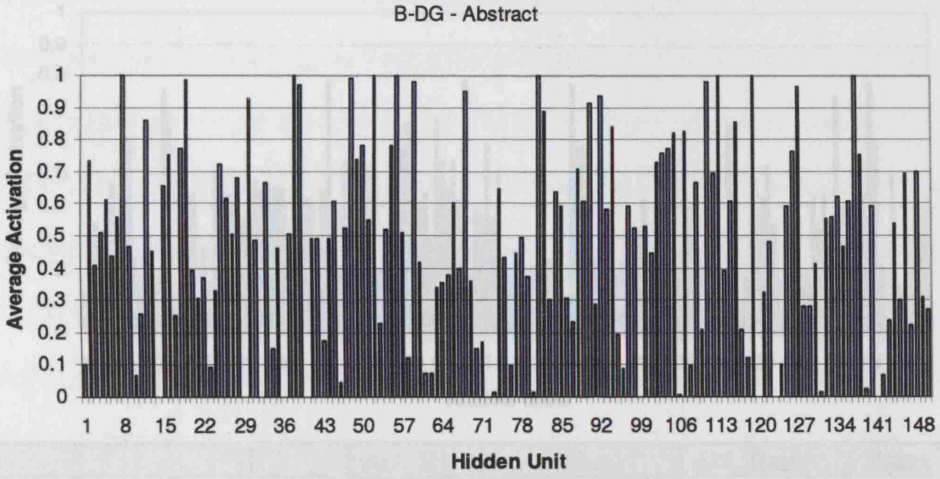


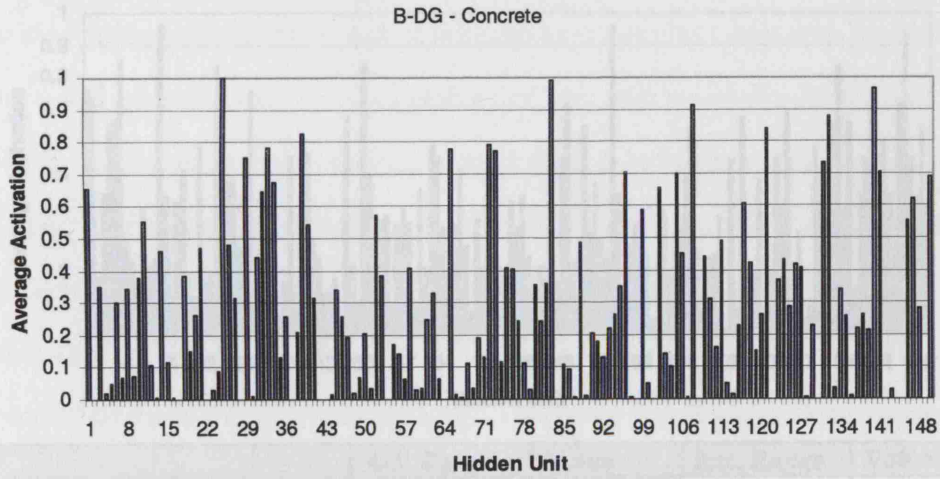

**Figure H.1: A simple three layer network showing how a hypothetical six-attribute vector might be presented to the network using the Glasspool and Plaut & Shallice approaches respectively. Red circles indicate ON values, white circles indicate OFF values, and orange circles indicate intermediate levels of activity. Histograms beneath each network show the sum of ON values for each attribute position produced by the Glasspool and Plaut & Shallice approaches for their respective 400 word corpora.**

Figure H.1 shows two three-layer feed-forward networks for (a) the B-DG approach and (b) the B-PS approach respectively. Each is indicative of the *types* of semantic inputs provided to a network using each design. Beneath both are histograms showing the sum of ON values for every attribute position across all corpus members, showing that the B-DG approach provides minimal contribution to the left side of the input vector. We cannot assume that weights from all input nodes are modified during the error correction stage of training since the error propagated back from the hidden layer to the input layer is multiplied by the input, and in the case of zero input, there is of course no weight change. Where this is applied consistently to the same input-to-hidden node connections, we argue that the network is effectively behaving as a partially, rather than the fully connected network it appears to be.

This is more clearly highlighted in Figure H.2 and Figure H.3 which show the activity of hidden units for abstract and concrete words for the Glasspool and Plaut & Shallice approaches respectively. The graphs show the average activation across 400 words, and the embedded tables highlight the proportion of actual activations between selected ranges. The tables show that for both networks, most hidden units are either very active (highlighted in yellow), or very inactive (highlighted in green). With the B-DG approach however, a hidden unit is slightly more likely to be inactive than active for abstract words (47.17% versus 42.61%). This is much more pronounced for concrete words where far fewer hidden units participate in a word's identity (69.04% versus 24.07%). A cursory examination of the graphs shows other differences between approaches. The Plaut & Shallice design shows qualitatively similar hidden unit activations with the activations of concrete words possibly appearing slightly sparser. For the Glasspool approach however, the average hidden unit activations for concrete words seem very sparse. In other words, there is a lot of white space between highly active hidden nodes. How then do we interpret this data?

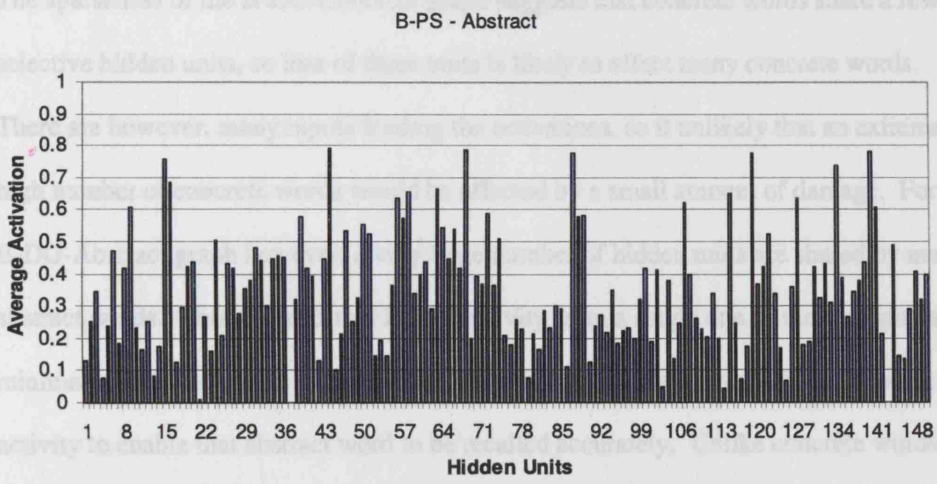



| | | Act. Range | Value | Act. Range | Value |
|---|---|---|---|---|---|
| | | **0.00 → 0.10** | **47.17%** | 0.50 → 0.60 | 0.93% |
| Average Activation | 0.48 | 0.10 → 0.20 | 1.95% | 0.60 → 0.70 | 1.70% |
| Standard Deviation | 0.47 | 0.20 → 0.30 | 1.21% | 0.70 → 0.80 | 0.50% |
| | | 0.30 → 0.40 | 1.12% | 0.80 → 0.90 | 1.89% |
| | | 0.40 → 0.50 | 0.91% | **0.90 → 1.00** | **42.61%** |

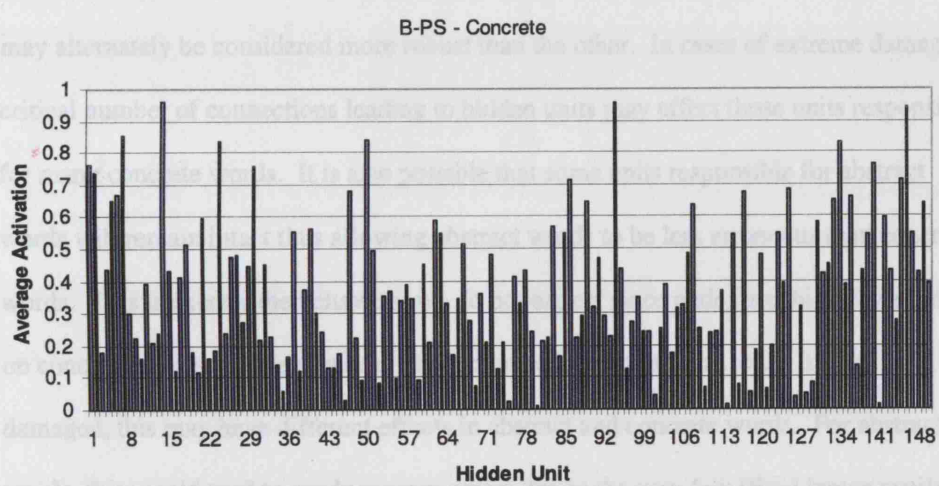



| | | Act. Range | Value | Act. Range | Value |
|---|---|---|---|---|---|
| | | **0.00 → 0.10** | **69.04%** | 0.50 → 0.60 | 0.59 |
| Average Activation | 0.27 | 0.10 → 0.20 | 1.35% | 0.60 → 0.70 | 1.29 |
| Standard Deviation | 0.43 | 0.20 → 0.30 | 0.93% | 0.70 → 0.80 | 0.17 |
| | | 0.30 → 0.40 | 0.71% | 0.80 → 0.90 | 1.18 |
| | | 0.40 → 0.50 | 0.65 | **0.90 → 1.00** | **24.07** |

**Figure H.2: The graph shows average hidden unit activations for a Glasspool Network across 400 words from a network trained with random seed 123. The embedded tables describe the actual (not average) activation ranges for all words across all units.**




| | | Act. Range | Value | Act. Range | Value |
|---|---|---|---|---|---|
| | | **0.00 → 0.10** | **57.67%** | 0.50 → 0.60 | 1.46% |
| Average Activation | 0.34 | 0.10 → 0.20 | 3.00% | 0.60 → 0.70 | 2.22% |
| Standard Deviation | 0.43 | 0.20 → 0.30 | 1.99% | 0.70 → 0.80 | 1.38% |
| | | 0.30 → 0.40 | 1.60% | 0.80 → 0.90 | 2.80% |
| | | 0.40 → 0.50 | 1.56% | **0.90 → 1.00** | **26.32%** |




| | | Act. Range | Value | Act. Range | Value |
|---|---|---|---|---|---|
| | | **0.00 → 0.10** | **60.58%** | 0.50 → 0.60 | 0.63% |
| Average Activation | 0.36 | 0.10 → 0.20 | 1.24% | 0.60 → 0.70 | 1.29% |
| Standard Deviation | 0.46 | 0.20 → 0.30 | 0.85% | 0.70 → 0.80 | 0.14% |
| | | 0.30 → 0.40 | 0.70% | 0.80 → 0.90 | 1.23% |
| | | 0.40 → 0.50 | 0.52% | **0.90 → 1.00** | **32.83%** |

**Figure H.3: The graph shows average hidden unit activations for a Plaut & Shallice network across 400 words from a network trained with random seed 123. The embedded tables describe the actual (not average) activation ranges for all words across all units.**

The sparseness of the B-DG-Concrete graph suggests that concrete words share a few selective hidden units, so loss of these units is likely to affect many concrete words. There are however, many inputs feeding the activations, so it unlikely that an extremely high number of concrete words would be affected by a small amount of damage. For the B-DG-Abstract graph however, a very large number of hidden units are shared by many abstract words. This suggests that loss of activity from a single one of these might have minimal effect since there are a number of *redundant* hidden units providing enough activity to enable that abstract word to be recalled accurately. Unlike concrete words, these hidden units are provided with relatively few inputs and the loss of any of these could be critical. Under the right circumstances, it is therefore possible that many abstract words are unstable in the presence of damage.

This provides us with the first explanation as to why abstract and concrete words may alternately be considered more robust than the other. In cases of extreme damage, a critical number of connections leading to hidden units may affect those units responsible for many concrete words. It is also possible that some units responsible for abstract words will remain intact thus allowing abstract words to be less erroneous than concrete words. This is exactly the behaviour we do not expect since patients exhibit fewer errors on concrete words. In the scenario where connections from input to the hidden units are damaged, this may have different effects in abstract and concrete words. For abstract words, this would tend to produce more errors due to the very few (five) inputs available to produce the expected activity in the hidden units. For concrete words, the high number (fourteen) of active input nodes may still provide enough activity for the words being spelled to be recalled better than abstract words, which is exactly the behaviour shown by patients.

For the B-PS approach, concrete words have slightly more active units (32.83% versus 26.32%) suggesting a higher level of redundancy for hidden units associated with

concrete words. In the presence of damage, it may therefore be possible to do without input from some of these hidden units retaining sufficient activity to recall a word correctly. This effect is more likely to be seen for concrete than abstract words, which is exactly the behaviour shown by patients.

We suggested that under the correct circumstances both abstract and concrete words may each be less susceptible to error in the presence of damage. We concluded this from observations made of hidden unit activations. Examining weights from the input to the hidden layer also highlights interesting behaviour. Table H.1 shows the average and standard deviation for the 4200 weights from both left and right sides of the input vector using the B-DG design. We show these values for three networks trained with different initial random seeds (123, 234, and 345). Histograms of weights from the left and right are provided in Figure H.4 and Figure H.5 respectively.

| | **Left** | | | **Right** | | |
|---|---|---|---|---|---|---|
| | **123** | **234** | **345** | **123** | **234** | **345** |
| Mean | -1.00 | -2.06 | -1.04 | -1.53 | -0.85 | -1.33 |
| Std Dev. | 9.53 | 8.03 | 8.91 | 7.53 | 8.59 | 6.74 |

**Table H.1: Average weights and their standard deviations for the 4200 connections from the Input to Hidden nodes for the B-DG network.**

Our first observation is that although average weights seem to be comparable, weights from the left show larger standard deviations suggesting the possibility of more erratic swings in hidden unit activation if these connections were omitted due to damage. We therefore analysed the weights in more detail identifying how many from the left and right had absolute values larger than 15.0.

| | **Left** | | | **Right** | | |
|---|---|---|---|---|---|---|
| | **123** | **234** | **345** | **123** | **234** | **345** |
| Mean | 1.12 | -7.61 | -1.15 | -11.13 | -1.18 | -14.29 |
| Std Dev. | 24.84 | 20.29 | 23.12 | 20.52 | 23.35 | 16.98 |
| N | 407 | 341 | 361 | 233 | 323 | 183 |
| **% of total** | **9.7%** | **8.1%** | **8.6%** | **5.5%** | **7.7%** | **4.4%** |

**Table H.2: Summary of statistics for weights from the Input to Hidden nodes for the B-DG network with an absolute value > 15.00. Weights from left-are clearly stronger than from the right. The % of total row shows the proportion of these relative to the total 4200 connections from each side.**

Table H.2 shows that weights on the left are consistently larger than those on the right.

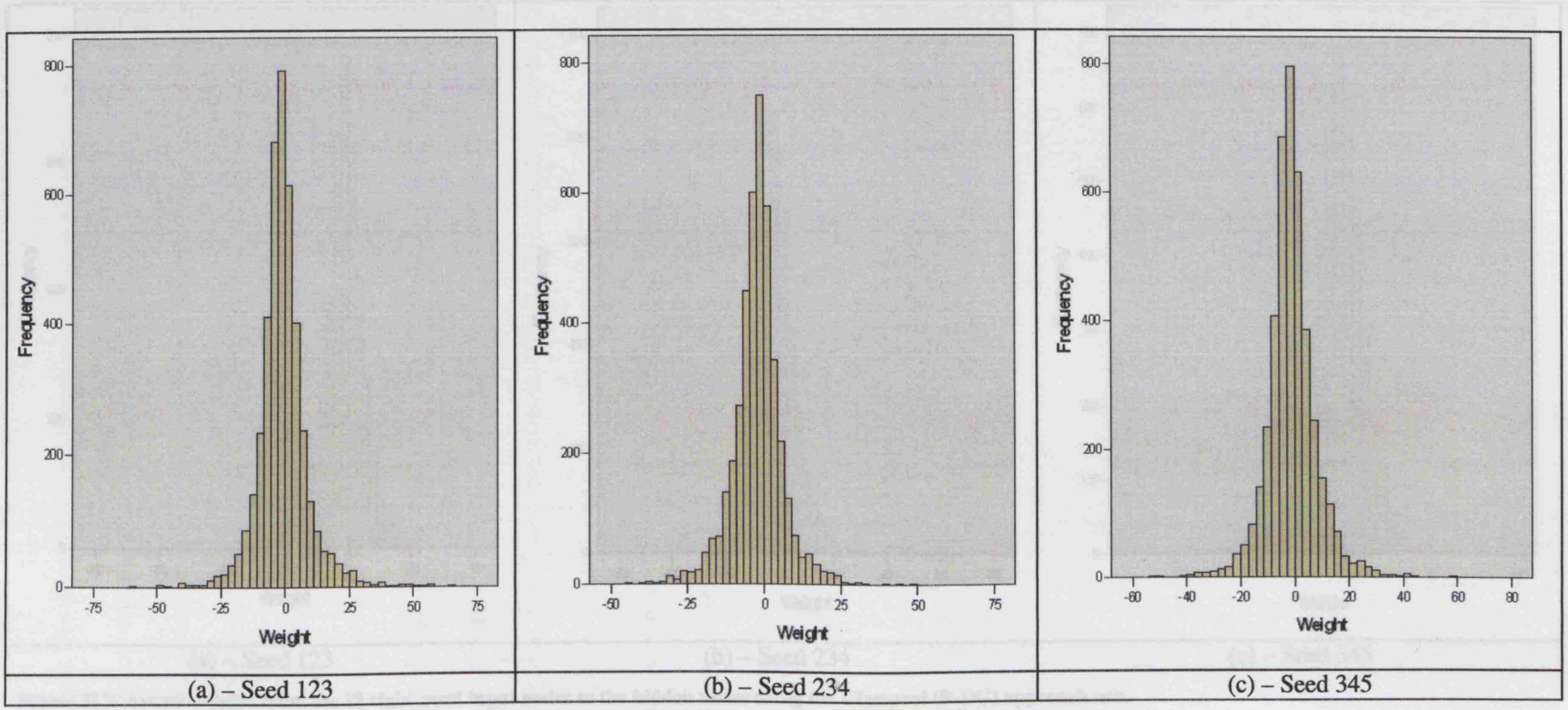
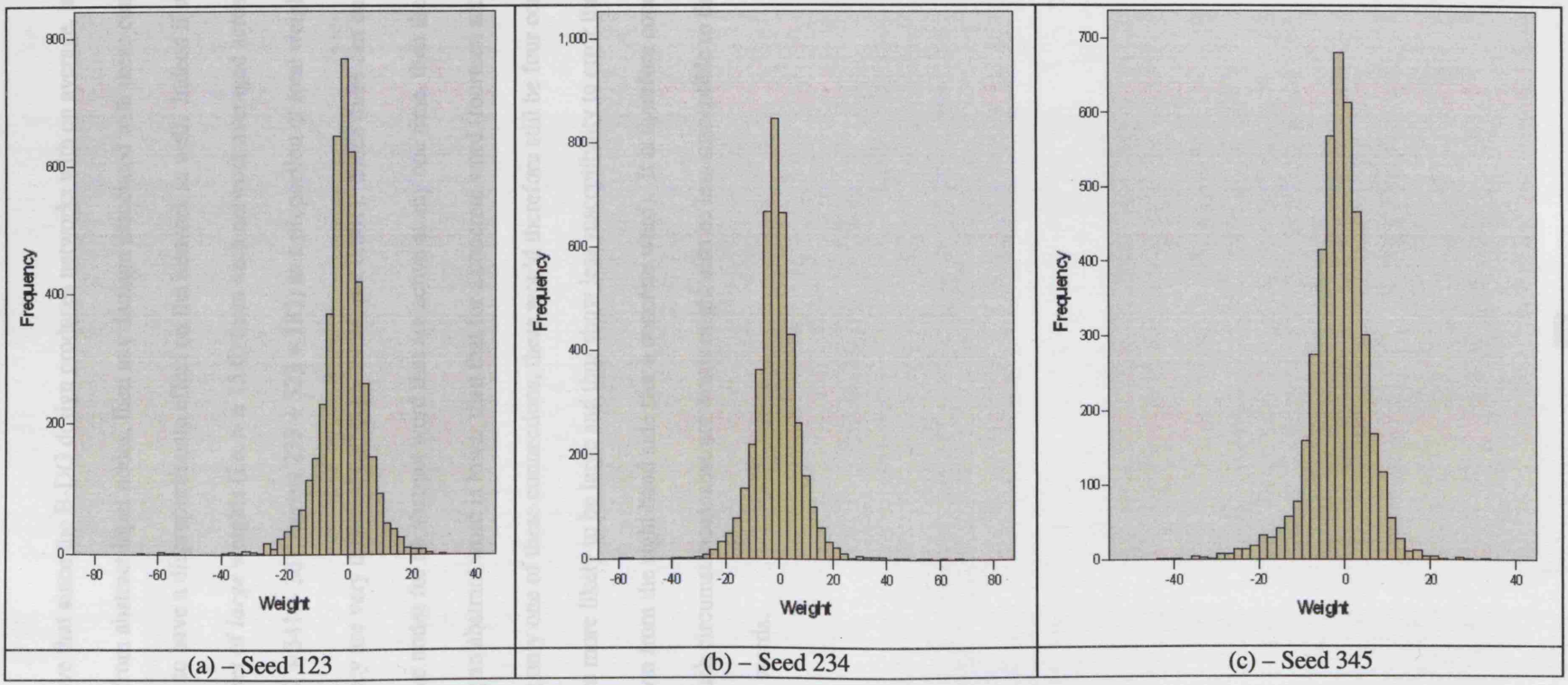



**Figure H.4: Actual weights from the 28 left-most input nodes to the hidden nodes using the Glasspool (B-DG) approach.**




(a) – Seed 123 | (b) – Seed 234 | (c) – Seed 345

**Figure H.5: Actual weights from the 28 right-most input nodes to the hidden nodes using the Glasspool (B-DG) approach.ach.**

We believe that since the B-DG design produces networks with on average, larger weights from abstract input nodes, then any damage associated with these connections will tend to have a disproportionate effect on the network as well. Indeed if we compare the number of *large* weights (i.e. $> \pm 15.0$) from each network averaged across random seed (407 + 341 + 361, versus 233 + 323 + 183) as a proportion of total weights (3 times 4200), they are very different ( $\chi^2$ (1) = 79.94, $p < 0.001$). Since there can be at most five active nodes for an abstract word that are active at any one time, then the chance of affecting an abstract word is lower than that for a concrete word (fourteen active nodes). In losing only one of these connections, there would therefore still be four connections, which are more likely to be large and thus show less susceptibility to error than were the connection from the right hand side (for a concrete word). It is therefore conceivable that in such circumstances, abstract words might also be less susceptible to damage than concrete words.

# Appendix I – Word Length Gradients

| Lesion Location | Lesion Type | Network A (% errors) | | | | | | | | | | | | | | Network B (% errors) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A-MW | | | | | A-IE | | | | | A-POS | | | | | B-GBDS | | | | | B-PSSB | | | | |
| | | Word Length | | | | m | Word Length | | | | m | Word Length | | | | m | Word Length | | | | m | Word Length | | | | m |
| | | 4 | 5 | 6 | 7 | | 4 | 5 | 6 | 7 | | 4 | 5 | 6 | 7 | | 4 | 5 | 6 | 7 | | 4 | 5 | 6 | 7 | |
| Random | Ablate | 39 | 51 | 59 | 65 | **0.09** | 51 | 58 | 63 | 70 | **0.06** | 37 | 50 | 61 | 68 | **0.10** | 58 | 63 | 66 | 67 | **0.03** | 45 | 52 | 53 | 56 | **0.03** |
| | Noise | 35 | 47 | 54 | 59 | **0.08** | 38 | 45 | 51 | 59 | **0.07** | 28 | 41 | 51 | 59 | **0.10** | 35 | 40 | 43 | 45 | **0.03** | 35 | 41 | 44 | 47 | **0.04** |
| | Constrain | 32 | 40 | 48 | 51 | **0.07** | 46 | 54 | 61 | 66 | **0.07** | 29 | 35 | 44 | 52 | **0.08** | 43 | 49 | 53 | 53 | **0.03** | 23 | 31 | 35 | 40 | **0.06** |
| | Scale | 27 | 32 | 38 | 40 | **0.05** | 32 | 41 | 51 | 58 | **0.09** | 17 | 21 | 29 | 37 | **0.07** | 19 | 31 | 38 | 40 | **0.07** | 10 | 18 | 20 | 27 | **0.05** |
| Input to Hidden | Ablate | 42 | 56 | 66 | 70 | **0.09** | 50 | 54 | 58 | 64 | **0.05** | 37 | 51 | 61 | 67 | **0.10** | 49 | 53 | 57 | 57 | **0.03** | 44 | 49 | 51 | 55 | **0.04** |
| | Noise | 27 | 40 | 48 | 52 | **0.08** | 34 | 40 | 46 | 53 | **0.06** | 26 | 39 | 49 | 56 | **0.10** | 38 | 42 | 46 | 46 | **0.03** | 34 | 38 | 40 | 43 | **0.03** |
| | Constrain | 27 | 39 | 48 | 51 | **0.08** | 39 | 45 | 54 | 60 | **0.07** | 24 | 34 | 46 | 54 | **0.10** | 58 | 61 | 65 | 64 | **0.02** | 24 | 30 | 31 | 35 | **0.03** |
| | Scale | 31 | 43 | 51 | 54 | **0.08** | 32 | 39 | 51 | 58 | **0.09** | 20 | 30 | 42 | 50 | **0.10** | 26 | 33 | 37 | 35 | **0.03** | 12 | 17 | 16 | 22 | **0.03** |
| Context to Hidden | Ablate | 46 | 60 | 69 | 74 | **0.09** | 62 | 67 | 71 | 75 | **0.04** | 46 | 58 | 69 | 74 | **0.10** | N/A | | | | | | | | | |
| | Noise | 25 | 36 | 44 | 51 | **0.09** | 38 | 42 | 46 | 52 | **0.05** | 33 | 46 | 57 | 64 | **0.10** | | | | | | | | | | |
| | Constrain | 29 | 41 | 49 | 53 | **0.08** | 39 | 41 | 46 | 51 | **0.04** | 22 | 28 | 37 | 41 | **0.07** | | | | | | | | | | |
| | Scale | 36 | 46 | 55 | 58 | **0.08** | 38 | 41 | 47 | 52 | **0.05** | 30 | 34 | 42 | 46 | **0.06** | | | | | | | | | | |
| Word Identity to Hidden | Ablate | 47 | 62 | 73 | 78 | **0.10** | 50 | 57 | 63 | 71 | **0.07** | 36 | 50 | 62 | 70 | **0.11** | | | | | | | | | | |
| | Noise | 33 | 45 | 53 | 57 | **0.08** | 47 | 55 | 60 | 67 | **0.07** | 26 | 38 | 47 | 53 | **0.09** | | | | | | | | | | |
| | Constrain | 17 | 35 | 49 | 60 | **0.14** | 31 | 37 | 45 | 51 | **0.07** | 19 | 38 | 54 | 66 | **0.16** | | | | | | | | | | |
| | Scale | 15 | 33 | 48 | 59 | **0.15** | 36 | 43 | 50 | 56 | **0.07** | 14 | 34 | 53 | 66 | **0.18** | | | | | | | | | | |
| Hidden to Output | Ablate | 38 | 51 | 62 | 67 | **0.10** | 54 | 62 | 69 | 76 | **0.07** | 32 | 46 | 57 | 64 | **0.11** | 41 | 45 | 46 | 48 | **0.02** | 36 | 44 | 48 | 48 | **0.04** |
| | Noise | 33 | 44 | 51 | 56 | **0.08** | 45 | 52 | 58 | 65 | **0.07** | 30 | 42 | 51 | 58 | **0.09** | 31 | 35 | 39 | 42 | **0.04** | 29 | 36 | 40 | 42 | **0.04** |
| | Constrain | 26 | 29 | 32 | 34 | **0.03** | 29 | 38 | 45 | 52 | **0.08** | 31 | 36 | 42 | 48 | **0.06** | 28 | 33 | 37 | 40 | **0.04** | 16 | 24 | 29 | 32 | **0.05** |
| | Scale | 47 | 50 | 50 | 47 | **0.00** | 20 | 31 | 44 | 62 | **0.14** | 32 | 33 | 36 | 43 | **0.04** | 21 | 27 | 33 | 39 | **0.06** | 18 | 28 | 37 | 44 | **0.09** |
| Output | Noise | 29 | 42 | 50 | 55 | **0.09** | 35 | 35 | 37 | 42 | **0.02** | 28 | 44 | 56 | 64 | **0.12** | N/A | | | | | | | | | |

**Table I.1: A comparison of network A and network B word length effects and their associated gradient. The above table shows the relative gradients calculated from the percentage of errors associated with each word length (four to seven letters), and a gradient (m) calculated using least-squares. The results from each network were based on the average of total errors for fifteen quasi-patients. A Mann-Whitney test showed that the gradients between networks A (N=63) and B (N=24) were significantly different ($p < 0.001$).**

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